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SECTION ON OPHTHALMOLOGY

PAPERS† AND DISCUSSIONS

THREE OPHTHALMIC QUESTIONS

OPTOMETRY, CONSERVATION, EDUCATION *

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BALTIMORE

Fellow Members of the Section on Ophthalmology:

My first and very delightful duty is to welcome you to this annual gathering—an event to which we have learned to look forward not only with pleasure but also with expectations of profit. Renewal of old friendships, contact with men whom we knew first for what they were doing and then claimed as personal friends, opportunities to blurt out our ignorance to hearers who are sympathetic because they have their own to swap for ours, shaking off for the time being the responsibilities and tire of office work and yet staying in the environment in which we are most at home—these are some of the things our annual meeting affords; and they are not lightly to be turned aside.

Some years ago I heard Dr. Osler say something like this—I am not sure even that he ever put it into print, but it ought to go there and ought to be remembered: "The doctor who lives to himself is a most dogmatic and dangerous animal. The patient watches his every expression; the nurse takes what he says as law and gospel; the family waits at the foot of the steps to get the latest news, and rejoices or trembles at the great man's verdict. After a while he gets to believe it all himself. Only when he touches elbows with his fellow doctors does he reach his true level, and because of this he owes it to himself and his patients not to forsake the 'assembling of yourselves together.'"

For my own part, I could afford to do without a great many things better than I could spare the help I get from an annual pilgrimage to this Section, and I know that I have plenty of companions feeling the same way. Of the social side of this meeting I can say only that I do not know anything about it. The Minnesota men served notice some months ago that they wanted hands off; but that provision is ample, and that the man who does not have a good time will have only himself to thank, are facts apparent already to those who know our hosts, and will be apparent to those who do not in a surprisingly short time.

I beg to thank you for considering me worthy of becoming your chairman, and for electing me to that position. I shall crave your indulgence for such errors as I may make, and for such success as attends our meeting I shall ask you to give the greater part of the credit to my friend who occupies the secretary's chair. Dr. Derby has been diligent in and out of season. In all the plans and work he has been ready with thoughtful suggestions and has always been willing to assume his own and others' duties.

He, Dr. Posey, chairman of the Executive Committee, and I decided to make an effort to secure some new working material for our meetings.

One cannot but be struck at society meetings with the number of men who are evidently doing good work, thinking about the problems presented in every-day practice, and yet taking no active part in contributing to the information fund. As the late Dr. Kipp of Newark once said to me, you get at what a man really thinks better on the hotel porch or at dinner than you do in a meeting. We have tried to get some of these men on our program this year, using those who have been the Section's mainstay in the opening of discussions. I think that we have achieved considerable success, and that the program shows not only new names, but also work well worthy careful attention.

In addition to those by members of the Section, we have three papers from invited guests. The trachoma question presents economic problems which have received careful study by the United States Public Health Service. We are indebted to the kindness and interest of Surgeon-General Rupert Blue for the detail of two of his officers, Drs. McMullen and Schereschewsky, to present this subject. With the addition of Dr. Stucky we have probably the three men who know most about the geographic, social and economic sides of this dreaded disease. The Massachusetts Commission for the Blind has sent Mr. Henry Copley Green, its field agent for the conservation of sight, to present to us the big problem of saving eyes; no one has had greater experience, no one can speak more authoritatively. Mr. Herbert E. Ives of Philadelphia has kindly come to discuss the subject of light. Finally, it has been our endeavor to emphasize the pathologic side of ophthalmology. A number of men working in pathology have brought instructive specimens, and will have time to

* Chairman's address.

† As the footnotes indicate, certain papers are here printed in abbreviated form.

demonstrate them. We have aimed to make the program short, but there is enough to require close attention to the end of the session.

OPTOMETRY

I ask your attention to a review of three matters of importance to the science of ophthalmology. The first is optometry. For years ophthalmologists have been forced to go before legislative committees and combat specious arguments in favor of elevating to a profession a large body of tradesmen. Our motive for opposing legislation desired everywhere, and now existing in thirty states, is conviction that it is the duty of a conscientious physician to oppose anything calculated to give the public false teaching on physical and medical truths. My purpose is not to repeat the arguments against such legislation, but to gather some lessons from this experience.

Optometrists have collected a not inconsiderable literature. All of us have read their articles. Refraction problems are discussed; quotations abound from standard medical works, and, on the surface at least, there is an appearance of scientific reasoning. All the way through, one can see a gradual approach to the "vital" element in refraction. I say "vital" not in the sense of essential—though it is really that—but vital in the sense of a living organism, with its circulation, nerve-supply, functions as an end brain organ; its power, on the one hand, to produce remote symptoms, and on the other to manifest evidence of distant lesion. Usually, as the optometrist writer approaches this point, reasoning wobbles. Soon we are in the familiar torrent of abuse of "drops," laudation of "fogging," attempts to draw conclusions from ocular phenomena clearly symptomatic, and so on. Evidence is conclusive that the writer is working with a mental bias.

For whom are these men writing? Are there in the optometric fold young, ambitious and conscientious men who are deceived into thinking that they are getting the real thing when they read optometric journals? Has the establishments of the optometry course at Columbia University attracted into optometry men with perfectly honest intentions, who believe they are entering a real science because a leading university is willing to take the apparent position of endorsing it? The question is not without importance.

There is, too, an entirely different class of literature, an example being a pamphlet called "Conservation of Vision and Modern Optometry" edited by the Publicity Committee of the American Optical Company. This pamphlet was widely circulated among physicians in Maryland before and during the session of the legislature of 1912. It contains, of course, the usual hysterical claims of glaucoma from "drops," without intimation of the existence of mydriatics which can be safeguarded. A striking feature of the pamphlet is a garbled quotation from the book of the late Dr. Roosa, published in 1889, about producing glaucoma. Omissions from the paragraph, as it appears in the book itself, add greatly to its value from the optometric point of view. Another stinking thing is an attempt to make the late Drs. Norris and Oliver advise against the use of mydriatics. A statement which appears in the book of these writers in the chapter on "Examination of the Eye" is used by the Optical Company under the caption "Danger of Instilling Drops." In the book there is not a word about "danger." The student is warned against relying on a dilated pupil for routine ophthalmoscopic examination. He is at the same time told to dilate the pupil unhesitatingly under certain conditions. All this is omitted in the Optical Company's pamphlet. One unfamiliar with the original source could form no other conclusion than that Drs. Norris and Oliver were opposed to the use of mydriatics.

Finally, just a mention of two items appearing in this May issue of the *Optical Journal and Review*. They are indicative of the mental state and intentions of optometrists. The leading article is entitled "Oculist Opposes Drops in Refraction." It is written by one Herbert Dickson, M.D., of Chicago. Dr. Dickson is evidently a newcomer into the Illinois metropolis, for his name does not appear in the American Medical Directory for 1910, but does in that of two years later. The article itself is a vapid effusion with a few well-known physiologic facts and practically nothing more, except a denial of permanent weakening of accommodation from "drops." This the editor criticizes in a foot-note, and naturally, for it is the optometric Gibraltar. An editorial calls attention to the "argument" against the use of "drops" by the "Chicago oculist," who, incidentally "expects to read a paper in July at the state optical convention in Chicago, explaining his method of treating cataract without operation, and showing how his treatment can be used in cases of glaucoma and optic atrophy." The doctor adds, "I am giving out this new matter at a meeting of optometrists rather than at a medical meeting because my practice has come to me through the help of optometrists and in spite of physicians, to whom therefore I feel no gratitude."

The other item has reference to the recent amendment of the New York optometry law prohibiting the title "Doctor" unless it has been conferred by a duly authorized college prior to the passing of the amendment. An editorial in the *Optical Journal* has this: "It is believed that a number of the optical colleges will be recognized so that graduates who receive degrees from them will be entitled to use the title." It is stated that the New York State Educational Department "gave assurances" to this effect. If I am right in my inferences, the optometrists' "scientific" articles have just enough scientific veneer to fool an uninformed person, student or legislator. Their writers abuse ophthalmologists, but emphasize and welcome every sort of seeming medical endorsement—even to the extent of manufacturing it by garbled quotations. They speak with editorial favor of scheming to circumvent an amendment to a state law forbidding them to use a title which has but one popular meaning, and which they have not earned.

These are the people we are called on to oppose in their endeavor to secure professional recognition. In this work we have done more than prevent legislation in a great many states. The necessary publicity of the matter has had an educational value. It is more generally appreciated now than formerly that glasses are therapeutic means for combating the results of congenital deformity, senile changes or functional results of disease, and that the diagnosis is part of medicine. This knowledge has spread to members of legislative bodies and it is rarely necessary to argue it.

If I correctly interpret the present situation, it is of social rather than strictly scientific nature. True, the latter is its underlying feature, but it is the former which affords the optometrist his greatest strength. I do not know that I can put it better than it was put to me by a member of the Maryland legislature, who has twice helped defeat optometric legislation. Substantially what he said was this:

The optometrist has only a small part of a big science; he is not trained to know even where his sphere of activity ends; his work is full of danger to the public, for without medical training he cannot be depended on to know the difference between health and disease; yet the fact remains that he often does give satisfactory spectacles, and there is a large class of persons whom he has served for years and will probably continue to serve until he makes some blunder. He asserts that he is trying to elevate his own standards.

Then I was asked, "Cannot the medical profession propose something positive?"

I bring this question to the Section on Ophthalmology, with another: Was it wise a year ago to abolish our committee on optometry? True, the *Bulletin* of September, 1911, brought the matter up to date and contains the basic information on which we must work; but legislatures continue to meet, and the situation is constantly changing. The plans of the Council on Health and Public Instruction of the American Medical Association, for broad study of all hygienic matters, put this and all health questions in a new light. I would not be understood as yielding to the present demands of the blatant commercial optometrists, totally unqualified for professional standing, even when judged out of their own mouths. Yet I feel that the Section on Ophthalmology has not done its last duty in this optometry business, and that we still have educational and probably legislative obligations to fulfil. I believe that the first step is the selection of a capable committee of five or more of our own members to take up the matter with the Council for public protection.

CONSERVATION OF VISION

With approval of the Executive Committee, an entire session is devoted to conservation of vision, the subject to be presented by Henry Copley Greene of Boston. Choice between the terms "conservation of vision" and "prevention of blindness" is not without significance. The former includes the latter, but it goes farther, and brings within our purview influences which affect eyes functionally, and which receive but scanty treatment in our own literature. It suggests means for bridging over the critical period of school life—means developed by the thought and skill of the illuminating engineer, the architect and other professional men. It brings us in contact with the social worker, the philanthropist and altruistic organizations.

About three years ago the Russell Sage Foundation held a series of meetings in New York, to which were invited representative men and women from different spheres of work. Much valuable information was gained. Prior to the national work of the Russell Sage Foundation, a committee of the American Medical Association had been collecting data on blindness, studying not alone immediate causes, but also underlying conditions. If one will glance over reports in our Proceedings he will find much of value on every medical phase of preventable blindness. There are, also, in engineering literature and in papers written by our own members, discussions of proper methods of lighting rooms, arranging school desks, home regimen, etc. Much suggestive writing also exists: What should be done for children with defective vision but still sight enough to enable them to obtain some education? What are the functions of the district nurse and the social visitor? How shall innocent children be protected from ignorant parents? These are but examples of activities opened by the concept of conservation. The ultimate goal is to unite into one all these forces, medical, social, educational, philanthropic, moving toward physical, mental and, indeed, moral conservation.

It was with this dream, if I am not mistaken, that men and women attended the Russell Sage conferences, and it was with this ambition that they entered into the formation of a National Association. And yet, with all this information at hand, with a great foundation ready to stand behind a strong movement for conservation, with thoughtful, studious men and women anxious to throw themselves into the work, we have, so far as practical organization is concerned, hardly commenced. Some things have been done in a few states; in some they have been done better than in others, and in most not at all.

In my judgment, national movements have accomplished so much less than was hoped for because work was started before proper foundations were laid. Of what use is literature unless there are arrangements to receive and distribute it? Of what use, beyond exciting a spasmodic and temporary interest, are exhibits, unless professional and public minds have been prepared for them, and unless there exist forces to push home their lessons? What is needed is the organization, in the medical ranks of every state, of a society to keep at work till the general profession has learned its duties, the philanthropist his, the legislator his. We need leadership. It should be furnished by this Section, acting with the Council on Health and Public Instruction of the American Medical Association. Dr. Posey has carefully studied this sphere of conservation. He, and Drs. Wood and Lewis, have been asked to present a report, which they will do at our executive session.

EDUCATION

In the educational number of the *Bulletin* of the American Medical Association, issued Sept. 15, 1909, there is published under Section 8 the report of the subcommittee on diseases of the eye, ear, nose and throat. The ophthalmologists on this committee were Drs. deSchweinitz, Jackson, Standish and Wilder. Among the recommendations made was that ophthalmology, in the sense of pathologic conditions, should not be taught during the first or second year; "whether the third or fourth year of the course should be selected for the presentation of these subjects necessarily is best decided according to the arrangements of individual schools." Before beginning ophthalmology, however, the student should have certain preliminary training "in the anatomy, structural as well as regional, of the eye." He should also receive careful training in its physiology "and an arrangement should be made with the department of physiology in each medical school . . . by which the students in the year preceding that in which they receive their training in these several branches should have the opportunity of obtaining the necessary physiologic instruction." There should also be clinical demonstrations to smaller sections of the class, the student being required to make his own examinations and diagnoses. There are recommendations regarding the number of hours of instruction, didactic and clinical, subjects to be included in the course, qualifications of the instructors, etc. The last section of the report contains this: "Thus far the report has made no reference to the instruction in pathology and pathologic anatomy. . . . In the number of hours allotted, a sufficient time must be given to this particular subject to enable the student to obtain a satisfactory understanding of his clinical work, and each teacher will

naturally include instruction in pathology in his course."

The recommendation that training in anatomy and physiology should immediately precede the study of abnormal conditions is important because the medical student of to-day has so many things to think about that he is in a more or less chronic state of mental acrobatism. If the superstructure is not put on before the foundation has had time to crumble, he can have only insecure knowledge; the farther his pathologic is removed from his anatomic and physiologic training, the less apt is he to understand the former or remember either. The same is true of the relation of a specialty to general pathologic principles: and here is a question for every conscientious teacher to ask himself—how well qualified is he to teach his own specialty on the basis of general pathology? Probably he received no such systematic training when he was a student twenty years or so ago; diseases were taught by symptoms, and treatment was based largely on the physiologic action of drugs. Do teachers sufficiently appreciate the change they must make in conception, and realize how readily the eye lends itself to illustration of general pathologic principles? It is no uncommon thing to find that our best students forget, after two or three years, the diagnostic signs of common eye diseases. The reason is not hard to find. They had not been given a proper thinking basis because the specialty had not been harnessed to pathology.

To determine the extent to which our medical schools have complied with these recommendations, I sent a questionnaire to the deans or the professors of ophthalmology of schools ranked in Class A plus, and A, as given in THE JOURNAL, Jan. 18, 1913. These questions included:

1. The year for preliminary instruction and position of the instructor.
2. Whether or not didactic instruction is given.
3. The time and extent of section teaching.
4. Elective courses.
5. Facilities for postgraduate work.

There are sixty medical schools classified as above, and up to the time I was compelled to prepare this paper, replies had been received from thirty-six. Probably a larger number would not materially affect conclusions.

All of the replies state that some instruction is given in anatomy and physiology, and in all but two of the schools prior to the teaching of ophthalmology. It is impossible to judge of the extent of this teaching. Evidently it ranges from mere presentation of gross anatomy to sectional laboratory instruction in both anatomy and physiology. Instructors are the professors of anatomy and physiology or their assistants in twenty-eight schools. In most schools this instruction is subsequently reviewed by the professor of ophthalmology. In four schools only the professor of anatomy is concerned, no mention being made of physiology. In two preliminary teaching is left to the physiologic department, and in two more it is given by the professor of ophthalmology in the first or second year. The time given to this preliminary instruction is:

First year only	4 schools
First and second years	16 schools
First, second and third years	1 school
Second only	10 schools
Second and third	2 schools
Third only	3 schools
Total	36 schools

The time relation between preliminary instruction and ophthalmology is:

According to recommendations of committee (i. e., in two successive years)	17 schools
One year between preliminary instruction and ophthalmology	11 schools
Two years between preliminary instruction and ophthalmology	6 schools
Two departments begin together in third year	1 school
Two departments begin together in second year	1 school
Total	36 schools

It thus appears that but one less than half of the schools heard from have adopted the plan of successive years; evidently there is a general tendency to follow the committee's advice. All of the eleven schools with a single year intervening end preliminary instruction in the second, and give ophthalmology in the fourth year. This is doubtless due to the crowded nature of the third year. Three schools attain the "successive" idea by giving special anatomy and physiology in the last part of the third year—a good idea if the chief course is put in the fourth, and if the third year affords time.

My second question had reference to didactic teaching. Evidently there is no stampede to do away with the old method. There seems to be some tendency to depart from lectures and to substitute reading, quizzes on this reading really taking the form of "conferences," "round-the-table talks," etc. The fourth year is still selected by the large majority of schools for didactic instruction. In my judgment this work should be put into the third year, when feasible, because it will receive more thought if introduced before the exigencies of graduation present themselves. Six schools neglect the didactic course entirely, or give it in a modified form. The character and standing of these schools make this tendency important. The University of Colorado, for instance, makes laboratory instruction a prominent feature of its didactic work. The University of California limits its didactic instruction to "a few lectures in the third or fourth year." Leland Stanford Junior University confines it to "association with clinical cases in the senior year." The reply from Cornell University states that didactic lectures illustrated by lantern-slides are given to the fourth-year students; the number of lectures is not stated. Evidently it is small. In Columbia, didactic teaching, if it can be so called, is limited to one clinical lecture a week for one-half year. The Johns Hopkins University gives no didactic teaching, apart from explanations in its clinical work, until the fourth year; then didactic lectures are elective. Required instruction in this university consists of ophthalmoscopy for two or three weeks in the second year and clinical demonstration daily for six weeks in the fourth year. The Iowa State University, the last of those showing a tendency to give up didactic teaching, seems to occupy a position of its own. A personal letter from Professor Dean illustrates how he has hypnotized the authorities of the university and obtained about all that one should desire—maybe a little more. He says:

> In the fourth year . . . about 50 per cent. of their time is devoted to ophthalmology. Throughout the fourth year the class as a whole receives two and one-half hours per week of instruction. This consists of clinical lectures illustrated by cases. Following the lectures the students see the cases, each case being then in charge of a separate demonstrator. The class is divided into three sections. Each section receives the following work for one-third of the year: three hours per

V. 6 1913

week of outelinic service; three hours per week of operative service; two hours per week of ward class work. During this time each student has at least six bed-patients under his personal observation.

In addition to this undergraduate work, Dr. Dean has at his disposal three salaried positions to which he can appoint graduates.

The wisdom of departing from didactic teaching is certainly questionable. It has to be decided according to the opportunities and environment at each school. Also the class of students is a factor. A good teacher, with students well grounded in anatomy, physiology and the principles of pathology, can no doubt save time and give more valuable instruction with the patient before him. On the other hand, there is a certain numerous class of students who will better understand what they see if they know previously what they are to look for, and on what symptoms and signs depend. Retaining or abandoning didactic teaching must be a local issue; but it is evident that there is a tendency to discredit it.

Section teaching is reported from every school. The preference is for the fourth year—twenty-five schools. In one school it is confined in the third year; in eight others it is divided between the third and fourth; and in two the year is not given—probably the fourth. A rather striking reply is from Cornell University; section teaching is limited to eighteen hours in the last term of the fourth year, a period, it seems to me, entirely too late to begin such work. Some of the larger universities have a certain number of beds set apart for eye patients; these are used in section instruction. It would seem that at least a beginning has been made toward providing such instruction as will lay a proper foundation for postgraduate work. The time devoted to section teaching varies from ten to forty-six hours per section. Comparing some of the reports with the catalogues, it is obvious that a practical difficulty in section teaching is obtaining a sufficient number of capable instructors. In some of the schools large classes have to receive this instruction from the professor of ophthalmology with one or two assistants. It is impossible with such inadequate force to meet the requirements of twenty hours' section teaching for each student.

As to elective courses, replies from twelve schools state definitely that there are none. Ten others recite clinical opportunities at general and special hospitals of which the student may avail himself if he so desires, but this is a matter of volition. In fourteen schools there exists some sort of an elective system. In seven of these it is limited to ophthalmoscopy and refraction. In one the student is allowed to elect ophthalmology, and if he so desires "spend all his time in this work." In but four of the thirty-six schools from which I received replies does there seem to be attempt to give elective courses in all departments. These are Johns Hopkins University, University of Colorado, Harvard and the University of Pennsylvania. The University of Iowa should probably be added to this list on account of the large scope of its work. The remaining school which reports an elective course is the Rush Medical College. I have placed it alone, because, coming as it does from so thoughtful a teacher as Dr. Wilder, it merits consideration as a standard for elective work. The course includes advanced work in anatomy, pathology and bacteriology of the eye, with courses in refraction. It seems to me that this limitation is wise. If the standards of ophthalmologic teaching and practice are ever to be elevated, the four subjects to which Dr. Wilder limits his elective courses must be the foundation. Unless men are tied up to these subjects, with, of course, needed clinical illustrations, they will not, save in rare instances, give them the necessary attention.

An interesting fact brought out by the replies is the small importance which teachers are attaching to operative courses for undergraduates. In but two schools are they compulsory, the Jefferson Medical College and the Medico-Chirurgical College. In the latter each student does six different operations on pigs' eyes. In two schools, Johns Hopkins University and the University of Michigan, operative work is elective. Reference is made in some of the replies to facilities for operating on animals' eyes if students so desire; but they are not encouraged to do so.

The Section has very recently heard about such postgraduate work as exists. The Universities of Pennsylvania and Minnesota are apparently earnestly engaged in organizing it on a scientific basis. Dr. Jackson's work in Denver is well known. The University of Iowa seems to have made real headway; Tulane University reports adequate opportunities for postgraduate hospital work, and Harvard has an organized course. Dr. Wilder, at Rush, offers what is essentially a continuation of the elective course to undergraduates. He says "facilities for postgraduate work embrace courses in pathology, anatomy, refraction work in the dispensary and at the hospital clinics." Three of my correspondents were good enough to outline their ideas of postgraduate work. For instance, Dr. Todd thinks that two years at least should be given to it; the first, as an intern in a hospital with time devoted to laboratory study, the second as an instructor and assistant in operative work; this, he thinks, should be followed by a year abroad. Dr. Jackson is in accord with Dr. Wilder regarding the necessity of strictly scientific branches; so, evidently, are many others.

It is impossible to divorce undergraduate work from postgraduate work. We recognize that a student cannot be made a competent ophthalmologist at graduation by any system available. But behind this truth there has lurked a tendency to reduce special teaching to what has been termed "essentials"; in other words, to attempt nothing more than teaching diagnosis of common diseases and how to avoid dangerous blunders. It will be hard to do more: *method of teaching this* is the important question.

As to past methods, we note that: first, many schools force the student to view ophthalmology as an addendum to, rather than an integral part of medicine—something to be taken seriously only if he expects to enter this specialty; secondly, the attitude of the schools has been reflected in state boards. Of fifty-three states and insular possessions, the laws of but seventeen require examinations in ophthalmology. In a few others a question or two sometimes appears on a surgical or obstetric paper, but that is about all. Without impressing the fact that every division of ophthalmology can be properly studied only from the point of view which controls all medicine; with the majority of state boards cutting eye diseases out of their requirements, is it any wonder that the six weeks' course has appealed to students as the logical thing? A few men—some of them recognized leaders in thought and practice—have tried to meet the situation by establishing postgraduate courses which begin with preliminary instruction in anatomy and physiology. We know the result. Students who thought beyond the standard of their undergraduate schools have created in such post-

graduate courses, in hospitals or private offices, opportunities for what Edward Jackson has termed "self-teaching." The rest, or a majority, obtained a veneer and became incompetent specialists, and state laws did nothing to prevent them. Introduction into undergraduate work of really careful preliminary teaching in anatomy and physiology, self-training in pathology by the eye teacher, if necessary, and ophthalmic instruction from these points of view may be depended on to change the student's mental attitude and fit him for postgraduate university opportunities. Through the proper channel in the American Medical Association, state boards should be urged to enforce the lesson.

Even then we must not lose sight of another thing. Ophthalmology is a very broad specialty. There is the relation between remote or systemic disease and ocular function or lesion; nature and reason of ocular manifestation; ametropia; relation of disorders of motility to remote troubles; differential diagnosis as to which is cause and which effect; the neurologic side of ophthalmology; its surgery; its all-important laboratory side; in a word, as Dr. Reber has pointed out in a recent paper, an oculist's office is almost a clinical laboratory in which special and technical investigations are made touching all parts of medical science. As we are advancing in our own thinking, so are our confrères in theirs. I question if we shall ever secure the symmetrically developed ophthalmologist, held up as the ideal by Jackson: a man trained and proficient, equally and adequately, in all these departments. The human equation and environment will always cause a certain amount of one-sidedness. Laboratory technic is very desirable, but it is not so important for the eye man as ability to deduct proper conclusions from laboratory findings, or to know when he needs laboratory help. He may be able to acquire this best from a laboratory confrère.

The first step in proper teaching of ophthalmology has been taken—insistence on preparation in undergraduate life for medical ophthalmic thinking. The second is to devise what Dr. Osler spoke of recently in Baltimore as "method—which is all we can teach students." We must remember that it takes time, and a good deal of it, to become fairly proficient in our own technical work; that we are preparing men for their life-work and they want, and ought, to get at it before their formative period has ended; that, after all, self-tendency will inevitably lead to self-teaching, and over-training has its dangers. Nothing is farther from my mind than a reactionary policy; but the essence of postgraduate work is neither multiplicity of subjects nor amount of time. It is drill in thinking: training in method. Simplification and formulation of that method is our present problem; and it is a most complex one.

With general professional awakening to the necessity of improved methods in ophthalmic education, and the response of schools to suggestions concerning undergraduate work, there should be no doubt of university response when asked to furnish adequate postgraduate facilities; but furnishing these facilities is only a beginning. Other avenues to ophthalmic practice will continue to exist. Even now there are here and there well-organized postgraduate courses, so far as they go. Capable and conscientious men, with no pretense to offering organized courses, take private students into their offices and hospital services, and see that training is founded on proper principles. Such teaching will continue. Unfortunately, so will the six weeks' courses and other attractive short cuts. In deciding where to take his postgraduate training many an honest fellow, with genuine desire to get the best and do his best, will be forced to consider the element of time. Periods of two or three years, after graduation, have been suggested as the earliest time at which a student be admitted to university postgraduate work. Add one or two years for this work, and a man may find himself confronted with the dilemma of giving up what he knows he ought to take, or entering on responsibilities which he knows equally well he cannot assume. Postgraduate work will develop itself, and experience will be needed to work out all mooted points. One item in method on which I think insistence should be made is that we avoid such detailed instruction as will, by its very length, hinder general acceptance. This is but one of the economic problems involved. Another is some regulation of state license for special practice. It is a matter of professional gratulation that at its meeting in Washington last month the American Ophthalmological Society appointed a committee to study this postgraduate problem with special reference to a degree of Doctor of Ophthalmology. Those on the committee are Drs. deSchweinitz, Risley, Weeks, Standish and Jackson. That a proper solution will be reached by such men may be accepted. In my judgment, this Section should have its own Committee on Ophthalmic Education, and I recommend its appointment, to report at our next annual meeting.

You may be interested in a criticism of our Section from Dr. Welch. He said to me, "The Section on Ophthalmology has been distinguished by two traits: it has kept out of politics and minded its own business." This business, he continued, has been to enlist the services of the best men in the specialty, to seek instructive papers, and to elevate standards. Can we formulate the purposes of our existence better than has this distinguished pathologist?

842 Park Avenue.

PHYSIOLOGIC OPTICS THE BASIS FOR TEACHING CLINICAL OPHTHALMOLOGY

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There are two features of our present methods of teaching ophthalmology to which I invite your attention. The first is that while the most important part of our subject is physiologic optics, we have made no adequate provision for teaching it. I have said elsewhere that physiologic optics is the most important department of ophthalmology because it is, on the one hand, to this that most of our time is devoted in our daily office practice, and because it is the one distinguishing feature which characterizes ophthalmology and which we do not share with other sections as we share anatomy, general physiology, pathology, materia medica, etc. But this understates the case. The fact is that physiologic optics so permeates the whole fabric of ophthalmology that it is as impossible for one to master any chapter of the subject without a good understanding of physiologic optics, as to weave a yard of cloth without the woof!

Let us consider a few chapters of ophthalmology with this in view. Take lacrimal troubles: Physiologic optics does not enter into the question of how to treat a lacrimal abscess or obstruction of the nasal duct, but that is not the whole chapter. How about the cases of lacrimation in which there is no obstruction to the flow of

V. 6
1913

tears from the conjunctival sac to the nose—in which there is excess of secretion? Here we must consider all the possible sources of irritation which may stimulate overproduction of tears, among others errors of refraction, also the conditions under which the eyes are used. This may involve investigating the character of the illumination, natural or artificial, the possibility of exposure to excessive light or the attempt to work with insufficient or improper quality of light. The mere fact that we are called oculists seems to imply that, in some way, we are endowed with the ability to decide these questions, though I am sure I do not know where instruction is offered to ophthalmologists in the important field of illumination; a field in which engineers are doing excellent work, but in which little is heard of ophthalmologists.

Consider phlyctenular troubles. In the majority of cases, occurring as they do in children, correction of faulty diet and hygiene and the application of yellow mercuric oxid or calomel will quickly cure. But that is not the whole story; there are cases, especially in older patients, in which attention to errors of refraction is needed to prevent frequent recurrence. Then, too, there are the problems arising from old scars of former phlyctenular ulceration — problems which of course involve physiologic optics.

Take the chapter dealing with the relations of the eye to diseases of other organs. When dealing with a patient referred to us under this heading we are liable to have to examine every part and function of the eye. Visual acuity must be taken, which requires the correction of possible errors of refraction and accommodation, and examination of the media, so that we can properly apportion any possible reduction of vision between defects of the optical part of the eye and defects of the retina, conducting paths and visual centers. Then the fundus must be examined, and of course ophthalmoscopy cannot be divorced from physiologic optics. Next there are the ocular muscles, including the pupil and ciliary muscles, which constitute a large chapter of physiologic optics. The visual fields have to be studied in settling many questions which arise in connection with diseases of other organs. Here is a chapter of physiologic optics which cannot be learned by intuition; a field in which we can ill afford to be ignorant of the work of others. The mere purchase of a perimeter does not make one an expert in detecting scotomas and mapping defects of the visual fields—still less in interpreting what he finds. Then there are questions of color-sense, central and peripheral; even the detection of dichromatism or abnormal trichromatism, though sometimes left to subordinate railroad officials, is not a matter that the mere possession of M. D. after one's name enables one to deal with in the absence of special training.

Enough; it is needless to take your time enumerating diseases and showing how, in one way or another, knowledge of physiologic optics is required for any mastery of the subject. How have we provided for teaching this subject any better than for teaching the less important parts of ophthalmology? We have not provided so well.

The subject of the teaching of ophthalmology in general has been treated by several writers recently, so that I need not rehearse that question. As to physiologic optics in particular there is this to be said: Most medical schools give some instruction, often with laboratory work, to first-year students in the course in general physiology. All postgraduate courses in ophthalmology give some instruction in physiologic optics because it is an essential part of every chapter in ophthalmology, but nowhere in this country has there been a regular systematic course in physiologic optics founded on laboratory work and covering all the main subdivisions of the subject in reasonably adequate fashion. This is a curious state of affairs when you come to think of it. It is just as if there were no course in pathology for students of general medicine, no laboratory work, no lectures, except what were given, incidentally, in connection with the instruction in all departments of clinical medicine and surgery of which it is an indispensable part, just as physiologic optics is an indispensable part of every department of clinical ophthalmology.

In discussing a proposed course to supply in some measure this lack, the question arose: Suppose a course of 300 hours, largely laboratory work, were offered in physiologic optics, who would take it? This brings me to my second point. In recruiting the ranks of ophthalmologists what method is followed? Something like this: a number of courses are offered in different branches of ophthalmology; any one who wishes to become an ophthalmologist is at liberty to look over the list, select what he thinks he would like, reject what he thinks he would not like or thinks may not be of use to him, and when he thinks he is ready he joins the ranks of the ophthalmologists. There is no one to criticize or pass judgment on his fitness, no one to assure the general practitioner, still less the general public, of his competence. Suppose that in the case of general medical education the courses of study were entirely elective, with no board to pass on candidates' fitness and grant them license to practice, what would be the consequence? Do not suppose that I am about to advocate a system of licensing oculists. There is a far better way. Provide an adequate course of training leading up to a degree, or diploma, as already proposed and begun by Dr. Edward Jackson and others, and the possession of the diploma will suffice for guarantee of at least minimum attainments in ophthalmology.[1] What I would plead for is a recognition of the basal, the supreme importance of physiologic optics in any adequate course of preparation for the practice of ophthalmology. Let an adequate course in physiologic optics be offered and, though the rank and file will doubtless pass it by if it is not required, the few who are not satisfied with attaining a "pass" grade, but want the best possible training within their reach, will, I believe, be glad to avail themselves of it. Should a preliminary course in physiologic optics be recognized by large eye hospitals as an important qualification in candidates for appointments as house-officers, a considerable step forward will have been taken.

Let us consider what should be included in our course in physiologic optics to make it "reasonably adequate." We would emphasize the value of paying special attention in the preliminary education to physics and of making mathematics cover algebra, geometry, and if possible some trigonometry. More mathematics can be taken to advantage, but cannot be regarded as essential. It is not merely that algebra, geometry and physics are used in physiologic optics; there is another reason for studying them, and that is the development in the student of some ability to look at a problem from the

1. Similar criticism can be made of the other specialties though the matter may not be so urgent with them as with us. There is no recognized standard of training and experience—of fitness in short—which a man need show that he possesses before posing as an expert in otolaryngology, in operative surgery, in gynecology, or any other special field.

mathematical point of view and to appreciate a mathematical proof.

Coming now to the course itself, what should be the scope and general plan of treatment? It would be wise to avoid making the work too exclusively laboratory, scientific and theoretical; rather the laboratory and the practical or clinical sides should be carried along together. This will be an advantage for the instructor, in preventing him from losing his practical grip, and an advantage to the student, in keeping up his interest and aiding him to grasp the subject and is, in short, the proper pedagogic principle. What we want is not merely to acquaint the student with the principal facts and laws of physiologic optics, but to make them so much a part of his mental equipment that he will apply them to all his clinical work—nay, rather, it will be so much a part of his mental atmosphere that he will see all problems through that medium, so that he will not have to *apply* the principles of physiologic optics to his work; his whole thinking and reasoning will be colored by, be in terms of, physiologic optics! This can be accomplished only by interweaving the laboratory and clinical work so that they never are divorced—never seem like two subjects, approached in two different ways.

Is this not clearly better than to have all the laboratory teaching done by one man, and all the clinical teaching done by others? The problem is not unlike the well-known question: which is better, a treatise on a subject by one author or one which is the work of several? Is not the answer that a single author is likely to produce a better introductory work as having more unity, consistence and proportion; while for advanced work a series of monographs by various writers may be superior as giving various points of view and permitting more exhaustive treatment, while the advanced student will not be misled, as a beginner would be, by contradictory statements made by teachers who hold different opinions on debatable points? The matter will probably be determined in any given case by the local conditions—what men are available and how much time they are willing to devote to teaching. If the introductory work has to be divided among several, then let the division be not into laboratory work by one and clinical work by another, but into subdivisions each treated by instructors from both the laboratory and the clinical points of view. After the introductory course there should be advanced work devoted to clinical or laboratory work, as the case may be.

If there is danger of the clinical work swinging away from the laboratory, there is also danger of the laboratory work getting out of touch with the clinical side—a tendency to devote too much time to problems solely of academic interest or having only a remote bearing on practice. It is not that I wish to disparage the study of pure science, as distinguished from applied science, but this course is not the place for it, and there are problems in abundance of every-day practical value, just as important scientifically and as suitable for training.

Another suggestion as to methods of teaching is that the student be required to solve problems in great abundance. We may well profit by the experience of teachers of engineering. If you will take the trouble to ask experienced and successful men in that work, they will probably tell you[2] that nothing gives a surer grasp of the subject, and a better mental training, than solving problems; it develops independence, self-reliance, accuracy, instead of loose, inaccurate thinking and dependence on rule of thumb. In connection especially with dioptrics and errors of refraction I would not only give students many problems (to be solved largely mentally, by the reduced eye of Donders), but I would require students to invent a great many for their own and each other's solution. The ability to state clearly what problem is presented by a given patient and, if possible, to reduce it to figures is a great help toward clear and accurate thinking, and is the best way to acquire the habit of using these methods in daily work.

Similarly, it is an advantage to have students make some of their own apparatus, or at least set it up themselves, since in this way far better insight is gained. There is a limit of course to the time that can be afforded for that work since, being an introductory course, it must cover a certain minimum number of topics.

Treated in this way it is at once apparent that the subject cannot be adequately covered in a few weeks. It is my opinion that from three hundred to five hundred hours is the minimum for a course designed to cover the essentials without trying to be exhaustive. The time required will depend a good deal on how much clinical work is included in this course and how much of that work is put into other courses. To be definite and concrete, let us apportion the time among the chapters without attempting to enumerate all the headings. For dioptrics and errors of refraction allow 150 to 250 hours. This must include: a review of physical optics; dioptrics of the eye; ophthalmometry; ophthalmoscopy; retinoscopy; hypermetropia, myopia, astigmatism, presbyopia; accommodation; mydriatics; and other allied topics. For ocular muscles and binocular vision allow 100 to 150 hours. This will include: anatomy and physiology of the ocular muscles; strabismus; insufficiencies; tests; stereoscopic vision, true and false; fusion training; projection; nystagmus; torsion, true and false. For the remaining chapters allow 100 to 150 hours. Here will be included: light sense, effects of radiation on the retina and other parts of the eye; adaptation; law of Weber or Fechner; sources of light; photometry; spectroscopy; distribution of light; lighting of houses, offices, schools, and other problems of illumination; color sense, normal and abnormal, central and peripheral; apparatus for detection of defects; visual fields for form, color, light; apparatus, sources of error, limits of accuracy; mind-blindness, optical delusions, and other psychologic problems.

The problems involved in teaching physiologic optics, on any such scale as above outlined, are so new to us that there is abundant room for discussion and differences of opinion. That there is room for difference of opinion as to the *need* of such training I am unwilling to admit.

2. Noyes, A. A.: Science, New Series, xxviii, 657. "It is power that counts; what a man can do, not what he knows. Problem-solving is by far the most effective means we have of developing mental power."

ABSTRACT OF DISCUSSION

DR. FRANK C. TODD, Minneapolis: Altogether insufficient attention has been given to the systematic training of ophthalmologists. It is therefore quite timely that the Section on Ophthalmology of the American Medical Association should consider the question of the proper preparation of the ophthalmologist. Dr. Lancaster is certainly correct in criticizing our present lack of system in such training, and his suggestion of the need of a more complete course in physiologic optics, which surely is an important basis for the teaching of clinical ophthalmology, is a wise one. If then we admit that he is correct in stating that physiologic optics should form the basis for the teaching of ophthalmology, it remains to determine the question as to the amount of time

that should be devoted to this subject. I confess that I do not feel able to determine this point. The beginner in the practice of ophthalmology will have little opportunity to perform the major eye operations, and I believe it is the usual experience of the beginner that he has no such operations for several years in his private work. Nearly all of the work that comes to him consists in the application of his training and study of the errors of refraction, muscle insufficiencies, or at least those diseases and afflictions dependent on such errors, so that in the average instance such a beginner would be better prepared to undertake the work he will be called on to do if he has had an adequate training in physiologic optics, than if he has spent a large amount of time in the preparation for operations which he will not have the opportunity to perform during those weary days of waiting. There comes into this discussion the question of the division of time and the relative importance of the various subjects that should be required to round out and properly train an ophthalmologist. It is a somewhat similar problem to that of determining the relative importance of the various branches of medicine and the amount of time that should be allotted to each department in the undergraduate medical school where men are being trained to become general practitioners. Indeed, it is a subject for careful consideration and thorough discussion, and I believe that most of us are as yet unsettled in our ideas, as we are only entering into its proper consideration. Thanks largely to the campaign that has been started by Dr. Edward Jackson, the profession and especially the teachers are becoming interested in this subject and alive to the necessities for a change. This particular question then is but one of many that should be settled in this very important matter of the proper and adequate preparation of an ophthalmologist. The scope of Dr. Lancaster's paper does not permit of the discussion of this larger subject at this time, and I hope it will not only bring forth a fruitful discussion, but that some action may be taken in the way of the appointment of a committee as suggested by our chairman in his opening address, to consider the whole question of the proper training of an ophthalmologist. Such a committee could prepare a model curriculum and make other recommendations that would be of very great value.

DR. EDWARD JACKSON, Denver: Dr. Lancaster's paper alludes briefly to the connection of physiologic optics with lacrimal disease. As he casually mentions it one might think it not an important matter, but it may be extremely important. A few months ago a woman of 30 came to me with this history. She had been treated for lacrimal obstruction for nine months by a man of wide experience, for many years professor of ophthalmology in a good medical school, and six and one-half months by a younger man, who has good work to show for a postgraduate course in Vienna. The lower canaliculus had been slit, solutions injected, probes passed daily, or at longer intervals. The lower lid and adjoining portion of the cheek were red and eczematous from the tears that ran over, although every night she used a protective ointment, without which the skin became much worse. She was wearing glasses R.+1 cyl. L.+1.25 cyl. axes 90°. She needed the cylinders increased 0.25 D., their axes shifted 15° and 8° respectively and +1. D spherical added. Without treatment of the lacrimal apparatus, she was markedly better in ten days. In a month she reported that not a tear had flowed over her cheek for over a week. In three months she was entirely well except that the lower canaliculus has been slit; and, as when I first saw her, entirely closed, probably from treatment. This is not the only case of the kind. Something like one-third of the cases that I see which have been treated for lacrimal obstruction are of this general character. They represent lack of fundamental training with regard to physiologic optics. Dr. Lancaster's remarks may be thought of as commonplace and unimportant, but they refer to facts whose enormous importance is not appreciated because they are so common.

DR. S. D. RISLEY, Philadelphia: My experience is entirely in accord with Dr. Jackson's remarks but I wish to say that in many cases I have seen persistent trouble in using the eyes for near work, due to retention of the tears, caused by a contraction of the puncta. When the tears are retained they accumulate along the border of the lower lid, forming a prismatic pellicle on the cornea, which, when the eyes are turned downward as in the act of reading, materially changes the refraction of that membrane. This condition is readily overlooked but when discovered is readily removed by dilating the puncta. Therefore, lacrimal disease may be a disturbing factor in physiologic optics.

DR. LUCIEN HOWE, Buffalo: Our attention should not be diverted from the main point at issue—the practical education of the ophthalmologist. The time has come when we can no longer cram the brain of each medical student with exactly the same group of facts. Instead of that we must make sure that everyone has a broad and secure foundation. Then in the last year we can allow each to select that group of studies to which he is inclined, or to which he shows special aptitude. If he prefers to know a little of each different branch as at present, he could receive, as he does now, the degree of doctor of medicine. But if, during the last year, he would study physiologic optics, the pathology of the eye, and do similar work in an ophthalmologic laboratory, these studies could be received by state law as an equivalent for obstetric operations, gynecology, radiography, or the minute details of dermatology, laryngology, or other specialties for which the ophthalmologist has no immediate need. A man thus specially equipped in ophthalmology might properly receive the degree, not of doctor of medicine, but doctor of ophthalmology, or ophthalmic surgeon, or be given some such title here just as he can now receive it, with even less special study, at Oxford. A similar evolution has already taken place in the colleges and universities with regard to other degrees. Those institutions no longer require each man to become a bachelor of arts, but with the same time devoted to equally good training in other departments, the student at the end of his four years is made a bachelor of science or an electrical engineer, or given some other appropriate title. If we have the pharmacist and doctor of dental surgery, why should we not also have doctors of ophthalmology? If that degree were granted by reputable institutions and approved by state law, it would better conditions, by graduating men better equipped than now and give us a better class of colleagues. Incidentally, it would be much worse for the opticians who now blind the public eyes in more ways than one, by calling themselves doctors of optics. I think the time has arrived for this next step in the evolution of the study of ophthalmology.

DR. A. EDWARD DAVIS, New York: Dr. Lancaster's paper and the Chairman's address certainly have a direct bearing on each other. We must not underestimate the importance of physiologic optics, for that is the superstructure. The basis and foundation should be the preparation of the student in the histology and pathology of the eye. In this country, at least, that is where the specialist is weak, and he is also weak in physiologic optics. We have to go abroad if we want to study the histology and pathology of the eye, and they constitute the very kernel of the subject, which takes us abroad more than anything else. We excel in this country in physiologic optics, although it is poorly taught. Before a man becomes a specialist, like the artist who paints the figure, he must study anatomy if he wants to make a good picture and put the drapery on correctly. If we train these men in the histology and pathology of the eye they are then ready to put the glasses on in the proper way. They know what they are driving at. Another point is to teach men the intimate relation between the eye and general diseases. An oculist should be something more and not something less than a doctor. In other words, where all have the same course to a great extent, a man should know his mind as to the line he will study later, before leaving the medical college; but failing in that, he must do it afterward. That is the time to get the superstructure. The disgrace of optometrists and optometry in this country is due perhaps in great measure to our own faulty training. No man should even be allowed to fit glasses unless he has all this training.

DR. FRANK ALLPORT, Chicago: I do not know whether I misunderstood Dr. Howe, but I judge that his idea is that the student in the latter part of his course in the medical college should elect what branch of medicine he is going to study, and fit himself for that. I do not like to allow that to go unchallenged, because I think we ought to bear in mind that the first thing essential to the education of a specialist is a thorough groundwork in medicine. No man should be a specialist unless he has first graduated in medicine and obtained the highest degree. We demand that the refractionists or optometrists shall not fit glasses because they are not doctors, then why should we take down the bars to somebody who seeks the higher education, it is true, but still who does not care to go through the grind of gynecology, obstetrics, etc., which he never intends to practice. These two courses are not compatible. If we demand a general medical education on the part of the refractionists and optometrists, we should also be willing to submit to the same dictum ourselves. No man, in my judgment, should become a specialist until he has first had a complete general medical education with all that that means, and has subsequently either practiced medicine for a certain number of years—I cannot say how long—in order to familiarize himself with the general principles of medicine and surgery, or, in lieu of that, has had an internship of eighteen months or two years in a general hospital.

DR. GEORGE H. PRICE, Nashville: I was much interested in that part of Dr. Lancaster's paper in which he gave the number of hours to be devoted to various divisions of the subject of ophthalmology. If the number of hours laid down in this paper are devoted to ophthalmology alone, it becomes the major subject in the fourth year of every student, unless there is special provision, as suggested by Dr. Howe, for the purpose of allowing the man who has reached the third or fourth year to specialize along certain lines. The trouble is —and it has come into the teaching in every school—that every man holding a chair believes his subject to be the most important in the institution. There is no branch or chair in any institution so important to the ophthalmologist as ophthalmology. We have not time in a four-year course to devote to ophthalmology the number of hours suggested in the paper. The suggestions made by Dr. Allport and Dr. Howe are good. The time is coming when we cannot finish the medical course in four years. Now we have to supplement it with the A.B. degree before students enter; and in time we shall give four years to the general course, which is an absolute necessity, and then there will be one or two years devoted to special work in each department, to make men competent to practice any specialty.

DR. CASEY WOOD, Chicago: In view of what Dr. Price has just said, may I ask Dr. Lancaster when he makes the statement that the study of physiologic optics should require three hundred to five hundred hours, whether he was talking of undergraduate or postgraduate study? It is not exactly clear to my mind.

DR. ROBERT SATTLER, Cincinnati: After spending a long time in the physiologic laboratories abroad and sharing at that time similar views to those so ably explained by Dr. Lancaster, concerning the necessity for better facilities of instruction in physiologic optics I returned with the determined purpose to afford advanced students in medicine and in particular those with a predilection for ophthalmology such advantages. I found, however, that this was an almost impossible task mainly owing to the passive interest and lack of preliminary training on the part of the student and after years of futile effort have since, with occasional exceptions, completely abandoned it as a purposeless undertaking. I advise every first and second-year man to take up the study of physiologic optics in connection with the work in physiology and have long urged that the hours devoted to the physiology of vision should be increased. I do not approve the teaching of physiologic optics to advanced students having no predilection for ophthalmology, but only for those who show a special bent, or who seek personally better opportunities for instruction in physiologic optics with the avowed purpose of selecting ophthalmology as a specialty after graduation.

DR. WALTER B. LANCASTER, Boston: I am sorry that the two subjects should become confused—the undergraduate and the postgraduate teaching. If Dr. Price would add to the three hundred hours allowed for physiologic optics, the number for pathology, etc., I think he would see the impossibility of it as an undergraduate program. My paper was written to apply to the training of specialists. When we have a program arranged as I hope it will be by a competent committee, we shall see that five hundred hours is much too short a time to cover the whole subject of ophthalmology. Two years, I should think, would be the minimum, and one-half year, under my program, might be devoted to physiologic optics. With regard to elective work for undergraduates after the second year, I am sure Dr. Howe does not mean after two years but rather three or four. We are coming to the five-year course. After three or four years devoted to general medicine a man is ready to do some elective work in his future specialty. The planning of the course in medicine has been a matter of accretion. A great many things have been added to the science of medicine in the last twenty or thirty years, every one of which has been added to the curriculum. The result is that it is overburdened and the work is not effective. We must begin the process of elimination, and when we do that we shall find that we can leave out many unimportant chapters to make room for the more important ones. We cannot escape the law pointed out by Dr. Jackson. "How much we shall know about one thing depends on how much we are willing to be ignorant of about others."

SOME MODERN VIEWPOINTS OF GLAUCOMA

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Recent additions to the literature of glaucoma contribute less to its varied and many-sided clinical expression, or to the acknowledged completeness of its pathologic anatomy, than to the mystery of its origin. It is not so much the long-discussed question whether glaucoma simplex belongs in a category of its own, where the disclosures of my own experience must place it; or even that glaucoma, and in fact all its acute inflammatory expressions, are caused by blood stasis; for that a circulatory obstruction is the exciting feature has been held and believed by many investigators since Roser's time.

The more modern point of view concerns itself with the questions: (1) if in chronic, simple or non-inflammatory glaucoma, the accepted well-known sclerosis and rarefaction of the vascular and protective tissues (that is, the increased tension), is the first and actual dynamic factor and subsequently, after an inexorable but more or less uniform course, develops or excites the glaucomatous impulse, consisting of at present unknown intraocular biochemical changes, which, if not arrested, eventually and certainly lead to slow extinction of function: (2) if in all acute or more or less clinically so declared cases (in which, however, it is unerringly recognized as acute inflammatory glaucoma), these assumed biochemical intra-ocular changes must be considered the first or real dynamic factors, subsequently causing pathologic changes of sclerosis and rarefaction, or similar tangible ones, in the uveal and protective tunics. In other words, have we really convincing proof of the identity of the two forms of glaucoma, other than the more or less uniform increased tension, which they have in common?

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

V. 6
1913

Both the glaucomatous process and increased tension, if excessive and short or unduly prolonged, lead certainly to blindness. There is a difference, however, that in the acute declarations, blindness is often coincident with sudden high tension and certain prodromal symptoms. Later on or after an acute or subacute attack, a more or less similar pathologic anatomy is discovered, identical with, or at least closely resembling that which is early, almost constantly and uniformly, present in the chronic expressions, which have been so minutely studied and are so thoroughly understood. In the acute disease again, higher tension is always present. In the chronic disease it is lower and in some cases even in doubt. On this latter point we must await the confirmation of an assumed fact by the more reliable scientific disclosures of statistical tonometry, now furnished us by the Schiötz and Gradle tonometers. We know that an eye which develops or grows in response to normal nutritional impulses must and can adjust itself, through the inherent elastic property of its own protective tunic, to moderate increase above its normal tension. In illustration of this, let me refer to the investigations of Leber and Uribe y Troncoso, proving that the eyeball of an otherwise normal individual can adjust itself even to increased volume of its contents. If this important inherent compensating property of the protective and uveal tissues exists, it may be the natural safeguard against transitory or even increased hardness and fulness from various circulatory causes and will overcome natural hindrances and minor superimposed ones which the incoming and outgoing currents of blood and lymph produce. Thus it may equalize or spontaneously adjust the former, permitting a more or less perfect equilibrium or normal tension to be conserved; adequate nutrition and normal function are by this anatomic expedient mysteriously and perfectly balanced and a normal tension is maintained throughout life.

If, however, through developmental aberrations (mesoblastic deflections) of fetal existence, the elasticity of the globe, which we can assume at this period has a greater compensating quality through increased or latent resiliency of its larger blood volume, is not preserved because new or accidental anatomic impediments have been added to the natural circulatory ones, as structural aberrations on the part of the mesoblastic or vascular structures, then it will follow that they or the effects they produce are or become impossible of spontaneous adjustment. The equilibrium, so mysteriously and perfectly preserved until then, without such accidentally added new anatomic variations, for an adequate blood- and lymph-supply necessary to unimpaired functions after birth, will be upset or interrupted. We can reasonably assume that just so long as this compensating property of increased or reserve elasticity holds out, though nutrition and future functional activity both may suffer, they need not be seriously impaired until the globe yields, as it must and will, to the greater opposing force of lymph and blood obstruction and retention. Owing to the thinness and delicacy of tunic tissues, during fetal existence, globe distention, more or less retardation of ingoing (blood and lymph) currents, longer retention of increased blood volume, impedimental obstruction from compression of the larger sinus outlets within the globe or at their bends into the vortex-scleral canals, will now come to pass as next to coincidental happenings and we will have, after birth, the typical anatomic and clinical living illustration of congenital glaucoma.

If, instead of developmental causes, those which belong to senescence, or more commonly to remote periods of senility, become the active, exciting agents which interfere with or completely upset that mysterious equilibrium of a normal globe—now no longer histologically the same—which we call or recognize as normal tension and for which we now have accepted standards or readings through the scientific precision of tonometers, there will be presented a clinical picture even more complete, in its consecutive, objective anatomic changes, than we are able to observe in congenital glaucoma. As we all know, we shall then have enacted before us an almost orderly succession of pathologic causes and their clinical counterparts characterized by inexorable slowness, undermining, consecutively and permanently, nutrition and function. The most important structure anatomically and physiologically—optic nerve and retina—may for a longer or shorter time after this have preserved a certain tissue integrity barely sufficient for the merest display of function. The complete loss of this is, in many cases, followed by secondary pathologic changes of confirmed atrophic degeneration.

We note, in such cases, how first the normal resiliency of the globe is sacrificed. This is followed, sooner or later, except in a small number temporarily arrested by appropriate medicinal or surgical treatment, by the loss, on the part of the protective and uveal tunics, of every compensating property the eye has. We may assign this, as Henderson does, to the uveal tunic, that is, sclerosis of the pectinate ligament; or with Priestly Smith to peripheric overgrowth of lens dimension and consequent contraction of the circumlental region; or, with Leber, to pathologic changes at the iris angle blocking the outflow of the lymph in or near the spaces of Fontana and Schlemm's canal. Or we might reach conclusions of a host of other conscientious workers, who have suggested equally plausible and anatomically proved local abnormal tissue factors, which they have embodied in simple or complicated theoretical speculations. Each is entitled to greater or less recognition, as all offer a partial explanation of a wide and complete pathologic expression of an obscure first cause.

It has long appeared to me that acute glaucoma belongs in a category of its own. It is an intra-ocular tissue riot precipitated by a sudden choking of the entry of its blood- and lymph-supply, which, if not speedily relieved, destroys irreparably the function of the eye. Only those who know its pathologic anatomy or are personally familiar with the furious, almost cyclonic, course of its clinical declaration, can have more than an inadequate appreciation of what the true acute glaucomatous declaration stands for. It is known that the sudden appearance of inflammatory glaucoma depends on many exciting causes, which spring up as senescence and emotional life-experience proceed, decreeing at the same time different tissue and psychic changes for each individual. In some individuals (predisposed to glaucoma from intra-ocular congenital causes of anatomic origin which have always been present, but previously unsuspected and undeveloped, until the necessary exciting moment) there develops a true or unmistakable explosion of acute glaucoma following such exciting cause. A spontaneous recovery, though possible before through the compensatory qualities of more or less normal (juvenile) tissue, resiliency of the sclerotic and its lamina cribrosa, the entrance openings of arteries and veins and similarly surmised ones of the uveal tissues, is now no longer possible. It may well be asked if the unmistakable attack of an acute glaucoma is

precipitated by the retention and acidosis of a larger volume of blood and lymph, in an assumed or actually more or less inelastic or sclerosed globe, with equally inelastic connective-tissue fabrics and senescent arteries and veins as well: and, with consequent deficient oxidation or other biochemical changes, if quasistrangulation of the globe is induced by congenital anatomic and acquired conditions as age proceeds, in consequence of this failure to absorb oxygen, creating, or directly responsible for the intra-ocular chemical changes which precipitate or usher in an acute attack.

One fact is established, that the beneficent achievement of surgery, the crowning professional discovery of Von Graefe, of a simple iridectomy made at the opportune time, executed with technical proficiency, will bring relief and save useful vision in most cases of acute inflammatory glaucoma, while its equally perfect execution with the greatest proficiency and the selection of the most opportune time will as certainly not accomplish this for chronic or non-inflammatory glaucoma.

Heerfordt's views, among the modern, are important because of his new anatomic discoveries pertaining to the vorticose sinuses of the chorioid and their intrascleral passage. In a complete and carefully arranged paper, Heerfordt states new anatomic facts and with amplified theoretical conclusions of older viewpoints (Stellwag, Roser) recites his own version of glaucoma. He defends the theory of Donders that glaucoma simplex is the prodromal stage of acute inflammatory glaucoma and asserts, accordingly, that two factors are concerned with the origin of the glaucomatous process, one being responsible for chronic or non-inflammatory and the other for acute inflammatory glaucoma. Furthermore, he assumes that non-inflammatory or chronic glaucoma depends on lymphostasis. He adds that lymphostasis rarely causes an increase of intra-ocular tension higher than 55 mm. Hg, and that it is definitely established that non-inflammatory glaucoma is arrested if an unobstructed outflow of lymph can be brought about.

He contends that the name "lymphostatic glaucoma" should be substituted for that of "non-inflammatory glaucoma." He holds to the view that the higher tension of inflammatory glaucoma cannot owe its origin to lymphostasis alone, but must have its own special cause and he finds this in the assumption that it is a hemostasis of the tunica vasculosa. He theorizes extensively on this point and adduces new anatomic facts which, more conclusively stated, are that the resulting hemostasis is brought about by folding or kinking of the walls of a larger vorticose sinus with compression at its internal point of exit and particularly within the lumen of its scleral passage or the vortex-scleral canal. He advances something new in the observation that the distended anterior ciliary blood-vessels formerly looked on as veins are in reality arteries. The following statement concludes his paper: "As so many recently discovered facts indicate that inflammatory glaucoma starts after hemostasis develops, as a complication of lymphostasis, it will be more correct to designate inflammatory glaucoma hereafter as lymphhemostasis, or, briefly, hemostatic glaucoma, for the reason that it bears the imprint of hemostasis."

But the really interesting and profitable disclosures in Heerfordt's article center in his description of the painstaking anatomic examination of a glaucomatous eye, the numerous sections of which actually furnish tangible proof of assumptions commonly held since Roser's time (1859), of compression and valve-like obstruction in the larger emissary veins of the uveal tract. His microscopic sections demonstrate what has long been surmised, that a kinking or creasing of the walls of the vorticose veins actually takes place. This part of his communication is a welcome message for it supplements what Czermak, Birnbacher, Elschnig and others have established by means of less complete technical methods. Even that which is histologically new and of adjudged value, however, leaves much to speculation.

The older point of view of Donders, that glaucoma simplex is the prodromal or incipient stage of an acute inflammatory declaration, is again brought out by Heerfordt and made part of his version of glaucoma. While my experience does not refute this conclusion, it has more often appeared to me that the more or less acute exacerbations, which so rarely happen late in the course of glaucoma simplex, took place in broken-down arteriosclerotic subjects and could be assigned rather to secondary local and general nutritional degenerative changes than to a long-deferred expression of an acute attack of true inflammatory glaucoma.

Both simple and acute glaucoma have long appeared ontologically alike, but each represents to me a distinct clinical and anatomic phase during the longer or shorter course of which the character of one or the other is preserved. After one or the other has reached a relatively advanced or confirmed stage, with extinction of function, I have then observed, in exceptional cases, that a heretofore simple non-inflammatory case or one clinically declared as such, would take on new characters as the result of secondary, local nutritional causes, largely through the local and unchecked inroads of senility. These characters resemble, in their subjective and objective aspects, acute or more often chronic inflammatory glaucoma.

Acute unrecognized expressions allowed to become chronic may also, after a subsequent invasion of nutritional changes, assume objective clinical characters strongly resembling simple glaucoma owing to the spontaneous reduction of tension, absence of pain and local congestion. It is commonly admitted that a hypertonic globe may, exceptionally, more often after it has reached its confirmed state (glaucoma simplex and subacute inflammatory) again become normal and even hypotonic. I have seen this in cases of chronic inflammatory glaucoma but no similar instance of glaucoma simplex has come under my notice.

Senile and even presenile states make inflexible encroachments on the connective tissues and vascular structures of the eyes, and, together with similar ones which precede or accompany them throughout the body, leave their unmistakable imprint and sound the dominant note as fundamental anatomic and etiologic factors of glaucoma in general and simple glaucoma in particular. Individual peculiarities, optical errors, emotional tendencies, developmental and acquired predispositions, decree for some a special clinical picture and for others a many-sided one (inflammatory glaucoma).

It has certainly appeared to me that the distinctive or natural characters of glaucoma simplex were conserved as such throughout its long courses and that those of acute and subacute inflammatory glaucoma were also so preserved, yet were, on account of their many-sidedness, mistaken or not recognized because of the frequent and necessary variation of their original characters.

Clinically the kinship of congenital and senile simple glaucoma appears probable although the congenital cases may seem strikingly atypical when closely compared with those of senescence. We should not wonder at this if we call to mind the necessarily obscure and remote inception dating back to fetal existence or earliest infancy. Senility and fetal life impart, the former rigidity (sclerosis); the latter, resiliency to a more or less elastic globe. It is quite reasonable to assume, at least, that senility as it advances shows greater resistance and earliest infancy a less resistance: that is, greater elasticity or stretching. We have the rigid, inelastic senile globe which Coccius, Stellwag and others assigned as a principal factor for the pathogenesis of glaucoma and we have equally positive proof of an excessively elastic or non-resisting globe in buphthalmos, which is the confirmed or absolute state of congenital glaucoma simplex.

The more uniform clinical picture and history of senile glaucoma simplex impresses this fact on me, that the discoverable pathologic changes of the eye grounds of circulatory retardation and obstruction are always in the foreground; and also, to a lesser degree, tissue rarefactions and sclerotic changes of the retina and optic nerve with similar changes of the central vessels suggesting reduction or even complete obliteration of caliber in their remote territorial distribution.

We can assume for most cases no other than a remote and obscure inception. We must acknowledge also that it is impossible to determine, even approximately, the length of time such pathologic changes as are noted have been in progress. But a larger insight and experience of the etiology and rational treatment of the disease may enable us to interpret intelligently and to glean a probable prognosis. We must carefully analyze, after eliminating other known causes for similar pathologic changes, in particular the more conspicuous ones of the nerve head, its color and depression, its central vessels, their caliber and light reflexes. And an even more difficult and uncertain task is to trace the vessels to their discoverable ramifications in the retina and from these ophthalmoscopic disclosures guess or infer whether the intercommunication between their arterioles and venous radicles is interrupted or retarded only. During a still more advanced progress, this inference is more certain for we can assume that the blood volume which passes through the central vessels is so minimized that it is sufficient for the barest display of useful function only, which can also be conclusively corroborated by perimetric and visual tests. The final or confirmed stage is reached amidst a painless progress, with fluctuating, moderate or greater tension, transitory betterment and failure of vision until its complete extinction is at hand. For a longer or shorter period after this, depending on the encroachments of age elsewhere and consecutive ocular ones, the structural but functionless integrity of the retina and optic nerve may be indefinitely preserved or, as not infrequently happens, such eyes may be invaded by secondary nutritional degenerations leading to a different clinical picture.

With a fuller knowledge of the pathologic anatomy and a larger clinical and surgical experience with glaucoma simplex, we must realize our helplessness, for even in spite of the most timely and carefully executed surgical treatment, most cases go to their doom. Many are even speeded toward a more rapid decline and others through the sudden and excessive relaxation of the operations (sclerotomy, iridectomy) lose, or suffer impairment of their already blunted central vision. We should all be governed in our advice for or against surgical interference by the mental and physical condition of our patient, even if functional tests still justify surgical interference, iridectomy, sclerotomy or its substitutes.

Gilbert, in an excellent paper, concludes his many practical and scientific generalizations with dependable advice as to surgical interference for glaucoma simplex. His paper also contains new facts pertaining to statistical tonometry and a just and fair analysis of Heerfordt's conclusions concerning the tonometric readings in so-called cases of lymphostasis and hemostasis. He also gives a lengthy account of venesection as a method of treatment during the prodromal stage of simple and inflammatory glaucoma, embodied in a carefully computed statistical report of cases observed at the Royal University clinic at Munich.

From my experience with glaucoma simplex, surgery is indicated only when an eye still possesses some of its natural or inherent compensating properties on the part of the sclera and uveal tunics. The main purpose of an operation, if these properties are not already lost, is to try to conserve and prolong them. If they are too much reduced or the globe is thereby too suddenly or too much relaxed, it will start secondary nutritional degenerations and, even amidst lower tension in some instances, more rapidly reach its final stage. Gilbert's advice on this point should be heeded by everyone whose experience with the various surgical measures which have been invented and practiced is limited, even though they may be indorsed and supported by common and individual experience.

The eyeball is tolerant of almost any carefully planned and executed interference, once it is in the grasp of advancing simple glaucoma, and it matters little whether the choice and judgment of an operator directs an iridectomy, sclerotomy, trepanation, cyclodialysis, iridotasis or an iridencleisis, if thereby he can only do a very little good; stay the final extinction of function and at the same time avoid doing more harm.

On the other hand surgery is as successful and beneficial now, for acute inflammatory glaucoma, as it was in the days of von Graefe and his followers. It is the same operation modified only by the acquired and natural proficiency of an operator. If executed in time on an otherwise healthy subject, in response to an unquestionable diagnosis, its next to successful result can be counted on.

If, after one or many subsequent subacute attacks, chronic glaucoma has been brought about through failure of the patient to seek competent advice or to follow that which was given, the operation, commonly attended by success, is more or less uncertain. When secondary or consecutive degenerations have invaded such eyes, surgery of every description sinks to the same level of ineffectiveness as is known to characterize it for simple glaucoma in its advanced course. For an acute attack not to be relieved through the right kind of surgery and at the right time, strongly suggests, with rare exceptions, that it was practiced on an incorrect diagnosis.

Though etiologically and pathologically it is not so different from other allied lesions, attended by higher intra-ocular tension, acute inflammatory glaucoma stands for me on a clinical territory of its own. It is through our own diagnostic shortcoming, however, that we become confused and confound a pseudoglaucomatous condition with a declaration of what only can or should be considered acute glaucoma. We have really not fully emerged from the benighted thought-period antedating the time of von Graefe and Roser. The latter, as is

known, took the ground against the former that glaucoma was primarily due to cyclitis and for this anatomic reason claimed that the more correct latter term should be selected for it.

If, as claimed, glaucoma was due to cyclitis, with massive hemorrhagic and lymph exudates into the vitreous followed by sudden high tension and pain, ascribed to compression or valve-like closure of the veins of the chorioid and retina (Roser), this contention should not, in the light of larger knowledge since gained, arouse surprise. From that time to the present we have been called on to differentiate just such cases of pseudo-glaucoma from the true glaucoma.

These conditions have only the cardinal symptom of high tension in common. This is transitory, however, and persists only until partial absorption of the massive exudates and hemorrhages into the vitreous and the anterior part of the uveal tract has taken place with release of pressure on the larger emissary veins of the uveal tract and retina, after which tension drops and pain subsides.

Except enucleation, which is not always expedient at once, surgical treatment commonly resorted to for the relief of high tension is of course unjustifiable and miotics either aggravate or act indifferently on the unalterable course which such affections take shortly after their inception.

Von Graefe, with a keener insight into the many-sidedness of clinical glaucoma and the broader viewpoint of a specialist, pointed out what was even then and certainly at present more fully known, that vitreous exudates and spontaneous rupture of retinal and chorioidal vessels is not a primary or even an infrequent happening during the early stage of glaucoma; he formulated and upheld his theory of serous chorioiditis with clear media and a visible fundus oculi.

If this point is clearly understood we can readily assume that a true attack of acute glaucoma may and often does take place in an eye without extensive intra-ocular vascular lesions. General cardiovascular lesions are present. Quite positively we can state that pseudo-glaucoma, in its clinical declaration, is almost akin to its true expression, and happens more frequently as the result of hidden or unsuspected degenerations of the circulatory system of the eye, especially of the ciliary body, and almost without exception as part of the expression of pronounced general cardiovascular and renal causes. Its declaration is as precipitous as that of true glaucoma but there is a different antecedent history for the attack. In the rush and haste to operate, as high tension and excessive pain seemingly demand or call for this at once, we have all had the unpleasant revelation that our diagnosis proved wrong and that we had resorted to just that surgical interference which must fail, as it affords the degenerated blood-vessels of the ciliary body a better chance to pour forth uncontrollable hemorrhage, which speedy enucleation alone can check. For such cases, with an unquestionable diagnosis beforehand, there is but one course to pursue and that is to temporize, to relieve pain by anodynes and later, if indications demand it, to enucleate such eyes.

Martin Fischer published, several years ago, a remarkable book on edema and another on nephritis. His edema theory aroused wide-spread interest, among our number in particular, for the reason of its direct application to glaucoma. In common with others my interest was absorbed as his reasoning at last appeared to me to point in the direction of physiologic chemistry for a solution of the origin of glaucoma. As special workers, we accept his "colloidal theory" and the statement that "the cause of edema resides in the tissues." Almost uniformly favorable recognition has been accorded to it by physiologists in particular and the scientific public in general. As specialists, however, we cannot accept his conclusions on glaucoma until he furnishes more conclusive proof for them than that contained in his writings and lectures.

In Chapter VII., on edema of the special organs, under the heading, "The Nature and Cause of Glaucoma," the following statement, among others, is noteworthy for its singular point of view: "The most intense grades of glaucoma can be induced experimentally in an eye by the absence of any circulation;" but we are informed farther on that this happens in enucleated eyes or after the death of an animal. We are told that an eye (enucleated) becomes glaucomatous not "because fluid is pressed into it but because through changes in it it absorbs an increased amount of water"; also that "the amount of such absorption is sufficient to explain the severest grades of glaucoma ever observed clinically," etc. His own version of glaucoma is fully outlined on page 120. He furthermore says: "The best evidence in support of this colloidal conception of glaucoma is, however, not furnished by mere hypothetical discussions but by the following clinical observations;" he also makes the statement that "glaucoma is in the eye what we call nephritis in the kidney."

But can we admit a parallelism, for an analogy is hardly tenable, between an important excretory organ like the kidney and an organ of special sense of the physiologic importance and dignity of the human eye?

Nevertheless we can confidently assume that Fischer is among those who are forecasting the dawn of new discoveries in physiologic chemistry which may follow and which will aid in the solution of the mystery which still surrounds the origin of glaucoma.

Seventh and Race Streets.

EXPERIMENTAL STUDY OF INTRA-OCULAR PRESSURE AND OCULAR DRAINAGE

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The large number of failures in the treatment of glaucoma may be attributed to only one cause: our ignorance regarding the nature of this disease.

When Mackenzie and von Graefe declared, half a century ago, that the essence of glaucoma lies in the increase of intra-ocular pressure, they proclaimed only a small part of the truth. The increase of intra-ocular pressure is a symptom constantly accompanying glaucoma, but it is not the essence of glaucoma. Even the zealous researches of Knies, Weber, Priestly Smith, Laqueur, Brailey and others could not advance much further the problem relating to the nature of glaucoma. The general trend has been and still is to detect the ocular changes which produce the increased intra-ocular pressure in glaucoma, but little has been done in the direction of investigating the general causes which bring about these changes.

In the light of newer methods of investigation and of newer points of view, the question of normal and glaucomatous intra-ocular pressure must be reviewed and reinvestigated.

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

Let us ask ourselves the following questions:

1. What is the relation of the intra-ocular pressure to the rest of the organism? Is there any relation between the nervous or circulatory system and the mechanism regulating the intra-ocular pressure? Is there any relation between the ductless glands and the mechanism regulating the intra-ocular pressure? Does the blood in glaucomatous patients contain substances which have something to do with the disturbance of the mechanism regulating the intra-ocular pressure?

2. Is there any reflex action, which, starting from some part of the organism, strikes the equilibrium of the intra-ocular pressure?

3. Is there any interdependence between the intra-ocular pressure of both eyes? Does it rise or decrease in one eye, if it rises or decreases in the other eye?

4. What is the rate of drainage of fluids in normal and glaucomatous eyes?

5. Do the extra-ocular or intra-ocular muscles have any influence on the intra-ocular pressure and drainage?

6. Is the local treatment of glaucoma logical, limited, as it is at present, to reducing permanently the intra-ocular pressure?

The solution of the foregoing questions involves such an immense amount of clinical and experimental research that it is impossible for any single investigator to cover the entire ground. This paper relates the results of a series of experiments and clinical observations which were made by me during the past few years, having as a guide some of the before-mentioned problems.

DIMINUTION OF INTRA-OCULAR PRESSURE OBTAINED BY A CONSTANT PRESSURE ON THE EYEBALL (OCULAR DRAINAGE)

It has frequently occurred that, when two ophthalmologists palpate the same eye, the first examiner finds the intra-ocular pressure high, while the other pronounces it normal. With the Schiötz tonometer, we have the same experience. If the tonometer is applied on the eyeball several times consecutively, the instrument registers at the end a lower intra-ocular pressure than at the beginning. Having this in mind, I have tried to find out what happens if the tonometer is kept constantly applied on the eyeball for from 30 to 120 or more seconds. I found that in normal eyes the intra-ocular pressure invariably decreases during the time the weight of the tonometer presses on the eye, and that this amount of decrease may readily be made out by simply watching the indicator of the tonometer moving to the right one or more divisions of the tonometric scale.

How can we explain this reduction of intra-ocular pressure produced by pressure on the eyeball by the weight of the tonometer for a certain number of seconds? How may we designate this phenomenon? In a previous paper[1] I used the term "ocular drainage," and explained it by saying that "the weight of the instrument expresses a certain amount of fluid from the eye, thereby reducing its hardness." Further: "If by applying a weight on the eyeball it is possible to express from this organ a certain amount of fluid, it is evident that there are channels in the eye through which this fluid is expressed." As is known the Schiötz instrument indicates the amount of intra-ocular pressure by measuring the depth of the depression produced in the cornea by the weight of the rod of the tonometer. This depression is admitted by Schiötz to be in inverse proportion to the intra-ocular pressure; namely, the higher the latter, the smaller the former. But a more careful consideration of this subject and the experimental evidence brought out by Wessely[2] shows that the data obtained by the use of the Schiötz tonometer are based on (*a*) the elasticity of the cornea and sclera, (*b*) the intra-ocular pressure—and to this I should like to add (c) the condition of the draining-system of the eye.

The degree of elasticity of the cornea is a factor to be taken into account in our measurements of the intra-ocular pressure, because the cornea is not equally elastic in all human eyes. Furthermore, Wessely has found that in the eyes of cadavers the cornea changes in elasticity rapidly, and unless Schiötz gaged his instrument on fresh specimens, he included in his findings an element of some error. The degree of more or less rigidity of the sclera has also some influence on the depressibility of the cornea.

Another factor to be considered is the patency of the channels for the inflow and outflow of ocular fluids. When the tonometer is applied on the eye and the cornea is depressed, it is evident that the intra-ocular fluid (the aqueous) being not compressible, is displaced somewhere. The fluid could be either driven further back in the eyeball, producing a bulging at the opposite side, or it could follow the path of outflow of the aqueous and drain outside the eyeball. The latter possibility, drainage, is the most acceptable. The following test seems to prove this:

Applying the tonometer on a normal eyeball for about sixty seconds, we see that the intra-ocular pressure decreases gradually. If, after removing the tonometer from the eye for a few seconds, we again measure the intra-ocular pressure, we still find it lower than that obtained at the beginning of the first examination. If the ocular fluid were not expressed from the eyeball by the weight of the tonometer, it would come right back to its original position when the tonometer is removed, and the intra-ocular pressure should be found the same every time it is measured. Massage of the eyeball, which is but another form of pressure, also reduces the intra-ocular pressure for a few minutes by expressing some fluid from the interior of this organ. Tonometric findings, therefore, include not only the actual amount of intra-ocular pressure, but also the elasticity of the cornea and patency of the channels of drainage of the intra-ocular fluids.

If we admit that the gradual diminution of the intra-ocular pressure following the steady application of the tonometer on a normal eye for from 30 to 120 seconds is due to an expression of fluid from the interior of the eye, it is reasonable to conclude that the amount of fluid expressed and the degree of diminution of intra-ocular pressure are in proportion to the weight applied and to the patency of the channels of outflow. In this paper are given a series of measurements of both intra-ocular pressure and ocular drainage, the latter term meaning the rate of diminution of intra-ocular pressure when the eye is subjected to the constant pressure of the tonometer for a certain number of seconds.

The technic of measuring the intra-ocular pressure, which is familiar to the reader, is the same in human and in animal eyes. For the measurement of the ocular drainage, the tonometer is kept steadily applied on the eye, while the assistant looks at the watch. As the indicator moves 1 mm. to the right, showing that there is

1. Schoenberg, Mark J.: Arch. Ophthal., March, 1913.

2. Wessely: Ber. ü. d. 38 Versamml. d. Ophthal. Gesellsch., Heidelberg, 1912.

a corresponding decrease of intra-ocular pressure, the examiner announces this to the assistant, who marks down the number of seconds it took the indicator to move from one division to another. The tonometer is applied for from 30 to 120 seconds. Lately, I have simplified my method by marking down how many divisions the indicator moves to the right during from 60 to 120 seconds. I then read from the tonometric chart how many millimeters of mercury equal a given number of divisions. A detailed description of the method of examination may be found in my former paper.[1] The plan pursued in this paper for the examination of the intra-ocular pressure and drainage is as follows:

1. Rate of ocular drainage in normal rabbits.
2. Experiments regarding the relation between the intra-ocular pressure of both eyes.
3. Experiments and clinical examinations relating to possible reflexes or action of distant organs on the intra-ocular pressure.
4. Action of extra-ocular muscles on intra-ocular pressure.
5. Ocular drainage in normal human eyes.
6. Ocular drainage in glaucomatous eyes.
7. Ocular drainage in eyes in which glaucoma is suspected.

I. OCULAR DRAINAGE IN NORMAL EYES OF RABBITS

Numerous examinations of the ocular drainage made on the same rabbit have revealed the interesting fact that measurements vary more or less at every examination.

In one rabbit at the first examination, the tonometer having been kept applied on the right eye for 295 seconds, the intra-ocular pressure gradually fell 9 mm. Hg (from 22 to 13). At the second examination it went down 20 mm. Hg in 540 seconds, at the third examination 9 mm. Hg in 385 seconds, at the fourth examination 6 mm. Hg in 420 seconds, and at the fifth examination 9 mm. Hg in 376 seconds.

The same kind of apparent irregularities in the measurements of ocular drainage was obtained in about ten rabbits. It seems logical to expect that the same tonometric weight, applied on the same eye, should reduce a constant amount of intra-ocular pressure in a given number of seconds, but this was not found to be the case.

The only constant factor in all the examinations made was that the intra-ocular pressure was reduced every time the tonometer was kept steadily applied on the eye for a given time.

II. POSSIBLE RELATION BETWEEN THE INTRA-OCULAR PRESSURE OF BOTH EYES

C. Hess[2] found in a curarized monkey that if an electric stimulus was applied on one eye, while cannulas connected with a manometer were introduced in the anterior and posterior chambers of both eyes, the intra-ocular pressure fell uniformly in both eyes and rose again after the stimulus was discontinued. No difference in the tension of the stimulated and non-stimulated eyes could be detected. Hess thinks that the change of intra-ocular pressure in both eyes was not due to the contraction of the intrinsic muscles, but to a change in the blood-pressure.

In another monkey the stimulation of one eye caused a slight increase of intra-ocular pressure in both eyes.

The ingenious experiments of Hess show that in monkeys it is possible to change the intra-ocular pressure of one eye by stimulating the other. I have studied the question whether there exists an inter-relation of intra-ocular pressure in the two eyes by measuring the tension in both eyes before and after massage or suction has been applied on one eye only and found that (1) the suction and massage reduce the intra-ocular pressure in eyes with a normal tension; (2) in eyes with a normal intra-ocular pressure the suction of one eye reduces very frequently the intra-ocular pressure of both eyes; rarely the intra-ocular pressure remains the same or becomes a little higher in the non-treated eye; (3) in glaucomatous eyes suction seems to have little influence on either the treated or the non-treated eye; (4) tests made on two cats under general anesthesia (ether) showed that heavy pressure and massage exerted on one eye does not change the intra-ocular pressure in the other eye.

IV. EXPERIMENTS AND CLINICAL EXAMINATIONS REGARDING THE EXISTENCE OF A REFLEX ACTION OF SOME DISTANT ORGANS ON INTRA-OCULAR PRESSURE

Although it is accepted as an established fact that in normal eyes the degree of intra-ocular pressure depends on the height of blood-pressure alone, and on no other factor,[4] I have tried to find out whether mechanical or electrical irritation of various organs in animals may change the intra-ocular pressure, and found that the mechanical irritation of the liver, spleen, kidneys, intestines and peritoneum did not produce any change in the intra-ocular pressure. If we noticed, during the experiment, a reduction of the intra-ocular pressure, it was due rather to the bleeding and shock accompanying the opening of the abdominal cavity. In the first and second cats in which respiration had stopped, the mechanical stimulation of the abdominal organs and the electrical stimulation of the brain could not elicit any reflex action on the intra-ocular pressure. Whether the regularly recurring increase of intra-ocular pressure after mechanical irritation of the sexual zone in the third animal was due to a real reflex or to a reflex contraction of the ocular muscles, or to a reflex vasomotor action of ocular muscles, I am not prepared to say.

I instituted similar examinations of human subjects. I had the good fortune to be allowed by Dr. A. A. Berg to measure the intra-ocular pressure in patients before and after various abdominal operations at the Mt. Sinai Hospital. Excisions of large portions of the stomach, intestines, of the gall-bladder, thyroid, kidney, etc., are more or less mechanical irritations to these organs and the intra-ocular pressure was examined before and after such operations with the purpose of establishing whether it changes during such operations.

TABLE 2.—SUMMARY OF CASES IN WHICH INTRA-OCULAR PRESSURE WAS MEASURED BEFORE AND AFTER OPERATION

Case	Diagnosis	I.-O. Pr. Before Operation O. D.	I.-O. Pr. Before Operation O. S.	Duration of Operation Minutes	I.-O. Pr. After Operation O. D.	I.-O. Pr. After Operation O. S.
9	Cholelithiasis	22	26	13	22	26
10	Fracture of humerus	30.5	30.5	15	36.5	36.5
11	Cholecystitis, lithiasis	22	....	20	30.5	....
12	Hernia inguinalis	22	22	10	26	26
13	Hernia femoralis	18.5	....	40	26	26
14	Chronic cholecystitis	22	22	15	36	36
15	Fracture of humerus	26	..	47	30	..
16	Carcinoma of stomach	22	22	68	26	30.5
17	Fecal fistula	26	26	58	26	26
18	Mesenteric lymphosarcoma	18.5	26	15	18.5	26
19	Acute appendicitis	26	26	10	30.5	26
20	Skin graft of leg	22	26	20	18.5	22
21	Carcinoma of stomach	16	16	15	18.5	18.5
22	Carcinoma of pylorus	22	22	48	18.5	18.5
23	Inguinal hernia	27	27	60	32	32
24	Exophthalmic goiter	30.5	22	70	44	32
25	Renal calculus	...	..	45	42.5	36
26	Iridocyclitis, O. D. enucleated	...	26	20	....	22

1. Hess, C.: Arch. Ophthal., xl, No. 2.

4. Leber: Handbuch der gesamten Augenheilkunde (Graefe-Saemisch), ii.

In all the adult patients, the anesthesia was begun with nitrous oxid for a few minutes and continued with ten minutes after the beginning of the anesthetic, and ether. The intra-ocular pressure was measured about at the end of the operation. I concede that this series of examinations is not free from objections. It may be noted as a known fact that ether anesthesia increases the intra-ocular pressure; the almost regular increase of intra-ocular pressure, therefore, which was found in this series of examinations, must be attributed to the administration of this anesthetic. The variations in the increase or decrease of intra-ocular pressure in these cases may be due to a fusion of several causes, as the variations of the blood-pressure during the operation, the changes in the function of the glands with internal secretion, nervous reflexes (?), etc.

In thirty-two eyes examined before the operation, three showed an intra-ocular pressure above 26 mm. Hg. (the normal upper limit); while after the operation (lasting from five to seventy minutes), the intra-ocular pressure was above the normal limit in fourteen eyes.

Considering the findings of my experiments on cats, examinations along this line must be made in a large number of cases before any conclusion regarding this question may be reached.

V. RELATION OF INTRA-OCULAR PRESSURE AND DRAINAGE TO THE TONUS OF THE EXTRA-OCULAR MUSCLES (MUSCULI RECTI AND MUSCULI OBLIQUI)

It is a well-known fact that external pressure on the eyeball raises the intra-ocular pressure. During the time the tonometer is applied on the eye, a slight pressure with the finger or a faint attempt to close the lids is sufficient to modify the tonometric measurement, and a high intra-ocular pressure is recorded.

The four musculi recti and two musculi obliqui, besides their main function of moving and rotating the eyeball in all possible directions, exert a certain pressure on the eye by the constant muscular tonus, which is inherent to a normally acting muscle.

Considering that the slightest external pressure on the eyeball could change the intra-ocular pressure, it is reasonable to suppose that extra-ocular muscles constantly act on the normal level of intra-ocular pressure by fluctuating its height within certain limits.

Wessely[8] has seen that in monkeys the intra-ocular pressure rises whenever the eyeball is moved. He found in a human eye with melanosarcoma before enucleation an increase of intra-ocular pressure of 5 mm. Hg every time the eye moved.

From a short perusal of some of the important contributions to the subject (Adamück, von Hippel, Grünhagen, Parsons, etc.) we may safely conclude that Leber's dictum "the intra-ocular pressure depends on the blood-pressure alone and on no other factors" ought to be modified. I am certain that the extra-ocular muscles have also something to do with the normal balance of the intra-ocular pressure.

My experiments on rabbits seem to corroborate the foregoing statement. In these experiments the intra-ocular pressure has been measured repeatedly on each rabbit; the insertions of the ocular muscles were severed, and the intra-ocular pressure and ocular drainage again measured the same day of the operation and on several days following cocain was used as a local anesthetic. The intra-ocular pressure fell considerably for several days after the tenotomies.

8. Wessely, K.: Ber. u. die 38 Versamml. d. Ophthal. Gesellsch., Heidelberg, 1912.

The decrease of intra-ocular pressure following the tenotomy of all the ocular muscles could be attributed to each of the following causes or to all of them:

1. The section of ciliary vessels and bleeding during the operation.
2. The action of the cocain and epinephrin.
3. The retraction of the ocular muscles and the temporary suspension of their tonic pressure on the eyeball.

The first two causes could not account for the persistence of the decrease of intra-ocular pressure for several days, since we know that the effect of cocain and epinephrin wears off in a short time, and the bleeding accompanying the tenotomies stops as soon as the conjunctival stitches are applied at the end of the operation.

VI. RATE OF DRAINAGE IN HUMAN EYES WITH NORMAL INTRA-OCULAR PRESSURE

If the tonometer is applied for from sixty to one hundred and twenty seconds on an eye with a normal intra-ocular pressure, we may see how the indicator is gradually moving a few divisions to the right. I have measured this gradual diminution of intra-ocular pressure in thirty-one eyes (some of them with optic atrophy, others perfectly normal) and found that in rabbits' eyes, the time required for a given amount of intra-ocular pressure to descend a certain number of millimeters of mercury is not the same in all patients. Furthermore the same eye does not show the same rate of diminution of intra-ocular pressure when examined at various intervals. For instance, in one patient, at the first examination the intra-ocular pressure of the right eye went down from 26 to 10 in 120 seconds, while at the second examination it decreased from 26 to 18.5 in 120 seconds.

The rate of diminution in thirty-one eyes is shown in Table 3.

TABLE 3.—RATE OF DIMINUTION OF DRAINAGE IN THE EYES OF FOURTEEN PATIENTS WITH NORMAL INTRA-OCULAR PRESSURE

No. of Eyes	Intra-Ocular Pressure mm. Hg.	Diminution Shown By Drainage Test: Maximum mm. Hg.	Diminution Shown By Drainage Test: Minimum mm. Hg.
2	26	16	7.5
3	22	6	3.5
3	18.5	7	5.0
8	16	6	5.5
8	13	8.5	3.5
6	11.5	5.0	1.5
2	10.0	3.5	3.5

Though the number of eyes examined is very small, Table 3 is of some value for orientation at least. I cannot say yet whether a certain given rate of diminution of intra-ocular pressure during 60 or 120 seconds in a certain eye is normal or abnormal because it does or does not correspond to the data in the table. For that purpose thousands with a normal intra-ocular pressure must be examined. For the present, I can only give my impression that the rate of drainage is smaller in eyes with a normal high (near 26 mm. Hg) intra-ocular pressure. There must be a minimum rate of ocular drainage for a normal eye. An eye which has less than that minimum rate is abnormal and most probably has glaucoma, no matter what its intra-ocular pressure may be.

VII. RATE OF DIMINUTION OF INTRA-OCULAR PRESSURE (OCULAR DRAINAGE) IN GLAUCOMA

Admitting that the gradual diminution of intra-ocular pressure obtained by the steady application of the tonometer is due to the expression of fluid from the interior of the eyeball, it is logical to suppose that in cases in which the rate of diminution of intra-ocular pressure

is smaller, the channels through which the intra-ocular fluid may be expressed are more or less obstructed. And vice versa, knowing that the entire pathology of glaucoma points to a partial or total obstruction of the paths of ocular outflow, we may reasonably deduce that our test will show a diminished rate of ocular drainage.

OCULAR DRAINAGE IN PATIENTS WITH ABSOLUTE GLAUCOMA AND GLAUCOMA WITH HIGH INTRA-OCULAR PRESSURE

CASE 41.—O. D., absolute glaucoma; O. S., aphakia, chronic glaucoma.

Ocular drainage: O. D. 86; no change in 120 seconds. After the use of pilocarpin drops for several days, O. D. 86; no change in 120 seconds.

As was to be expected, in all the cases of this character (nine) the intra-ocular pressure did not recede when the tonometer was kept constantly applied on the eyes for from 60 to 120 seconds. The obstruction in the drainage system in eyes with absolute glaucoma (angle of anterior chamber, canal of Schlemm, vorticose veins, etc.), is advanced to such a degree that no fluid can be expressed by the weight of the tonometer.

VIII. OCULAR DRAINAGE IN EYES IN WHICH GLAUCOMA IS SUSPECTED

This patient did not complain at all about her left eye on her visits to the clinic. The first examination of the left eye revealed that the ocular drainage was at a minimum as compared with ocular drainage of normal eyes with the same intra-ocular pressure (table of ocular drainage in normal eyes). The fact that the intra-ocular pressure remained at 18.5 while the tonometer was applied on the left eye for fifty seconds made me suspicious of the condition of this eye. That the suspicion was well founded was proved by the second examination, just before an iridectomy was performed on the right eye. At this examination, both the high intra-ocular pressure and the obstruction of ocular drainage confirmed the diagnosis of latent glaucoma. It is very important to mention that the left eye developed symptoms of acute glaucoma a few weeks after the iridectomy of the right eye.

CASE 65.—L. B., aged 65 years. O. D., absolute glaucoma. O. S., suspicious glaucoma. During the two years the patient was under observation, she complained of occasional blurring of vision in the left eye. Examination revealed nothing abnormal.

August 6: Ocular: O. D.

March 2, 1912: Ocular drainage: O. D. 60; no change after 50 seconds. O. S. 26. After 10 seconds, 22. *After another 65 seconds, no change.*

April 10, 1912: O. S. 18.5. After 60 seconds, no change.

Several more examinations of the ocular drainage of the left eye gave similar results: a relative impairment in the rate of ocular drainage, and though the examination of the fundus of the acuity of vision, and of the field of vision (roughly taken on account of the extreme nervousness of the patient) together with the normal intra-ocular pressure, were against the diagnosis of glaucoma, I could not regard the eye as normal on account of the reduced ocular drainage. Jan. 22, 1913, I was called to the patient's home and found an acute glaucoma of the left eye. Intra-ocular pressure was 42.5. V.= movement of hand. Cornea steamy, pupil immobile and dilated. This condition confirmed my former suspicion that the patient's left eye was in a stage of prodromal or rather latent glaucoma, all the time during which she was under observation, and that the relative impairment in the rate of ocular drainage came as a danger signal long before the intra-ocular pressure rose high above the normal limit.

CASE 66.—J. H., aged 36 years, nurse, came to the clinic of Mount Sinai Hospital, Feb. 9, 1912, complaining that she had suffered from headaches for about three months and that the pains were most severe around the left side of the forehead and temple. She sometimes saw various colors around the gas-flame.

Ocular drainage: O. D. 22. After 15 seconds, 16. After 60 seconds, no change. O. S. 26. After 60 seconds, 22. After 60 seconds, no change.

The rate of ocular drainage in these eyes did not seem to be quite normal. After two weeks, during which time she was using pilocarpin drops in the left eye, a second examination of the ocular drainage was made: O. D. 16. After 35 seconds, 13. After 25 seconds, 11.5. O. S 22. After 65 seconds, no change.

As we see, the rate of ocular drainage remained abnormal in the left eye, and the intra-ocular pressure did not decrease very much under the influence of a miotic.

At several examinations of the left eye at various periods, we found a lower capacity of drainage and a slightly higher intra-ocular pressure than in the right eye.

From my point of view, the fact that the intra-ocular pressure was found constantly higher in the left eye than in its partner, and also the circumstance that the rate of ocular drainage was partially impaired, spoke in favor of glaucoma.

If any conclusion is to be drawn from four cases of suspicious glaucoma it is the following: *The diagnostic value of a so-called normal intra-ocular pressure is only relative.* In these four cases the intra-ocular pressure was found normal, and still two of them developed symptoms of acute glaucoma. Is the impairment of ocular drainage of a diagnostic value, independent of the height of the intra-ocular pressure?

The following case should serve as a proof of the relative independence of the ocular drainage and the height of intra-ocular pressure.

CASE 67.—M. L., aged 38 years, cutter of cloaks. Both eyes, simple glaucoma; O. S., coloboma iridis artefactum.

June 1, 1912: O. D. 50.5, O. S. 30.5. As the use of pilocarpin, 2 per cent., 1 drop four times daily, did not reduce the intra-ocular pressure, the patient was directed to instil the miotic every hour in both eyes.

Examination of the ocular drainage, seventeen days later: O. D. 27; no change during 120 seconds. O. S. 22. After 32 seconds, 18.5. After 72 seconds, 16.

As we see, the miotic succeeded in reducing the intra-ocular pressure in both eyes to a degree "within the normal limits," but it did not act in the two eyes in the same manner. In the left eye both the intra-ocular pressure and the ocular drainage were normalized, while in the right eye, the ocular drainage remained impaired although the intra-ocular pressure became normal.

SUMMARY

The study of the ocular drainage has revealed the following:

1. There is always a gradual reduction of intra-ocular pressure if the tonometer is applied on a normal eye for a certain number of seconds.

2. The rate of reduction of intra-ocular pressure varies not only in various eyes, but also in the same eye if taken at different periods.

3. Experimental evidence seems to indicate that changes of intra-ocular pressure in one eye may often be followed by similar changes of intra-ocular pressure in the other eye.

4. Neither the experiments on rabbits and cats nor the examinations in the operating-room give any clue regarding the existence of a reflex or biochemical action starting from some distant region and influencing the intra-ocular pressure. The extra-ocular muscles play an

V. 6
1913

important rôle in the various normal fluctuations of the intra-ocular pressure.

5. The ocular drainage in glaucomatous eyes differs from that of normal eyes. The slower the rate of drainage, the nearer the eye is to an acute attack or to absolute glaucoma; the more rapid the rate of drainage, the nearer to a state of compensated glaucoma. *A reduction of the rate of ocular drainage may mean latent glaucoma in spite of an intra-ocular pressure which is within the normal limit (below 26 mm. Hg).*

The continuous fluctuations in the intra-ocular pressure and ocular drainage in normal eyes, the relative dependence of the intra-ocular pressure on the general blood-pressure, and of the latter on the ductless glands, and the probable relation existing between the intra-ocular pressure of both eyes, suggest that the present tendency of devising all possible operative procedures for the relief of intra-ocular pressure in glaucoma is only a palliative measure. It is not logical. The essence of glaucoma is not an increased intra-ocular pressure just as a high blood-pressure is not the essence of arteriosclerosis.

I wish to thank Drs. J. Maller and D. Halle for their assistance in my animal experimentations and Drs. J. Mills, O. Schirmer, H. Tyson and A. A. Berg for their kindness in permitting me to examine also some of their patients at Dr. Knapp's New York Ophthalmic and Aural Institute and Mount Sinai Hospital.

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HYDROPHTHALMOS

WITH A HISTOLOGIC REPORT OF TWO CASES, ONE OF WHICH PRESENTED A CONGENITAL COLOBOMA

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In the spring of 1912 there occurred in my clinic at Wills' Hospital that curious coincidence occasionally noticed by every clinician, of seeing within a short period of time several cases of an uncommon affection. In this instance it was hydrophthalmos, and the coincidence was even more striking in that all three cases were in negroes.

As I could recall seeing but six cases of this disease and as four of these occurred in negroes, in two of which there was a strong suspicion of hereditary syphilis, it seemed as if this might be more than an accidental association. With this thought in mind and with the recollection of an unfortunate outcome to the operative treatment by different methods and by two different operators of the eyes of a baby presenting a marked degree of hydrophthalmos, it was believed that it would be of value to collate the opinion of others as to the etiologic relationship of race and syphilis to this disease, and also their experience in the treatment of this condition.

The unusual opportunity of making a histologic study of an eye with a congenital coloboma which developed hydrophthalmos presented itself and the findings though incomplete are deemed of sufficient interest to publish.

SYMPTOMS

A brief description of the affection will here be given in order to recall to mind the conditions to be taken into consideration in a discussion of its treatment. Hydrophthalmos (buphthalmos, congenital glaucoma) is a condition due to intra-ocular hypertension probably arising from anomalies of development or of prenatal inflammation in the anterior segment of the eyeball. The striking contrast between the effects of hypertension in the juvenile eye and the adult eye is to be explained by the greater elasticity of the tissues of the young eye.

Some evidence of the disease is usually present at birth or makes its appearance within the first six months of life. Collateral and direct heredity has been traced in some instances. The disease is more often bilateral than unilateral and occurs with somewhat greater frequency in males. It is characterized by a uniform distention of the globe. The cornea becomes greatly enlarged and thinned and usually cloudy; dark lines appear in the hazed area due to tears in the membrane of Descemet. The anterior chamber becomes very deep because of the increased size of the cornea and the recession of the lens and iris. The iris is often tremulous and may be rent, more often at the ciliary attachment. It may be rudimentary or present a coloboma. The distention of the perilental structures causes the suspensory ligament to stretch and tear and thus is brought about a partial or complete luxation of the lens into the vitreous or anterior chamber. The lens is either normal or undersized. The sclera becomes thinned and appears bluish because the underlying chorioid and ciliary body show through. Notwithstanding the decided lengthening of the axis of the globe, the degree of the myopia is usually not very high, indeed occasionally the refraction is hyperopic. The chorioid and retina become thinned and atrophic and the optic disk cupped. If the hypertension is not reduced the distention of the globe becomes extreme, the eye becomes irritable and painful and may rupture spontaneously; or because of its protrusion and amaurosis, it is liable to injury and is ruptured as the result of a blow or ulceration. In some instances the tension diminishes spontaneously and the distention of the globe is permanently arrested. This usually does not take place until the process is advanced and the eye blind, but occasionally it occurs early enough for the preservation of useful vision. Among the more unusual associated conditions and complications are plexiform neurofibroma of the lids, congenital anomalies either of the eye or other parts of the body and ulceration of the posterior surface of the cornea secondary to iridocyclitis. At times the haze of the cornea is the first symptom to appear and leads to the belief by some that it is also the initial pathologic change, resulting in an anterior uveitis culminating in a secondary glaucoma.

ETIOLOGY AND PATHOGENESIS

Schmidt-Rimpler sees no reason to assume that hereditary syphilis exercises an influence in the origin of hydrophthalmos although he points out that, in the parenchymatous keratitis of congenital syphilis, hydrophthalmos does develop and states that a large percentage of Seefelder's cases occurred in families in which diseases of various kinds resulting in death in childhood was the rule.

There is much the same controversy here as in glaucoma of the adult relative to cause and effect of the various anatomic changes found. In one of the most recent and exhaustive anatomic studies of infantile glaucoma, Magitot concludes that there are two classes of cases, one in which there is little or no inflammatory lesion, including very few cases. These show an obliterating endophlebitis, of the emissary scleral vessels and some of the anterior ciliary veins. When such lesions are absent there exists an aplasia of the venous system of the angle, and Schlemm's canal is absent. Perhaps all our other congenital anomalies are due to some infection or irritation transmitted through the maternal placenta

The other class of cases is characterized by inflammatory lesions; these are common but of variable intensity. The slightest cases affect the venous system of the angle, then the retinociliary region and chorioid become affected, while in the most intense cases the whole of the uveal tract and the vorticose veins are attacked. He believes that the vascular inflammatory lesions are the cause of the increased tension in infantile glaucoma, and that it is not necessary to suppose an obstruction to the outflow of the aqueous humor. He suggests the existence of a posterior glaucoma from obstruction to the vorticose veins and an anterior glaucoma from that of the emissary ciliary veins.

Schoen believes the cause to be a congenital absence of the meridional fibers of the ciliary muscle which in the normal eye, by their elasticity, intercept the intra-ocular pressure from immediate impact on the elastic sclera. Heine, having found a striking thinning of the anterior scleral zone, concludes that there is a deficient resistance at that point, and Abadie likewise locates the primary cause there in the form of a retinochorioiditis. In connection with the views expressed by these two authors it should be borne in mind that Erdmann in experimental glaucoma of rabbits found a primary atrophy of the chorioid and retina exactly limited to the anterior segment of the eye due to the increased pressure. Von Hippel believes it to be due to a fetal disease of the cornea. Collins' views are based on the study of several hydrophthalmic eyes. He found in some cases a prenatal condition of the ligamentum pectinatum—a network of fibers filling up a space at the angle of the anterior chamber, which in the normal fully developed eye is part of the anterior chamber—and to him it seems probable that the persistence of this prenatal state of the ligamentum pectinatum is responsible for obstructed exit of fluid from the eye, and the occurrence of increased tension. De Lapersonne espouses the theory of Angelucci and Galleinga which is predicated on a congenital disturbance of circulatory equilibrium of the interior of the eye. He has observed in the vasomotor ataxia of Cohen, whatever may be its cause, a vasodilatation with hypersecretion explaining all of the symptoms of hydrophthalmos; and has actually seen cases associated with the vascular disturbance of Basedowian type. A like observation is attributed to Angelucci. Reis, who made a pathologic study of seven buphthalmic eyes, found no pathognomonic constant finding. In four of his cases the angle of the anterior chamber was open; in two of these there was complete absence of the canal of Schlemm. Parsons concludes that, whatever be the exact mechanism, it would seem certain that there is a very definite obstruction to the filtration of lymph from the eye at the angle of the anterior chamber. Whether this is due to a congenital arrest of development or to an intra-uterine or an early extra-uterine inflammation, must, in his opinion, be left an open question. He adds that even an arrest of development must have some cause, and that this is most probably to be discovered in some intra-uterine inflammatory or toxic condition. Magitot has expressed the same thought. Stimmel and Rotter describe the conditions found in three buphthalmic eyes studied by them. In the first case the canal of Schlemm was entirely absent; the ciliary muscle had a fetal structure and the iris and ciliary body were abnormally developed. There was also rupture of Descemet's membrane, hemorrhages into the retina and chorioid, detachment of the retina, old thrombosis of the central vein and recent thrombosis of the inferior vorticose veins. The other two cases showed absence of the canal of Schlemm, rupture of Descemet's membrane, chronic iridocyclitis and slight atrophic excavation of the optic papilla.

They state that the consensus of opinion is that the disease is caused by an absence, entire or in part, of the canal of Schlemm, which is placed abnormally far back; in the persistence of temporary fetal connective tissue in the angle of the anterior chamber, and in an insufficient separation of the iris from the cornea.

TREATMENT

The proved value of iridectomy, in glaucoma of the adult, has undoubtedly been the reason for its selection by many operators in treating the congenital type. Yet the anatomic conditions and the topography of the parts are so different in the two types that what is a safe and satisfactory procedure for the one becomes a dangerous and disastrous procedure when performed for the other. This operation has the support, however, of a number of surgeons of large experience. Thus Hirschberg reports a case in which sixteen years after the operation tension was normal. There were macular changes, a flat excavation of the nerve-head and a high myopia.

In Schoen's interesting case in which iridectomy still controlled the process after twenty years a cystoid cicatrix had resulted, the cyst being almost as large as the globe.

Schoenemann reports seven cases permanently arrested. He does not consider the operation dangerous if performed early and after the method of Schweigger.

Schmidt-Rimpler evidently does not share this view. He quotes von Graefe to the effect that "in the vast majority of cases the disease remains a *noli me tangere* and adds that he has unfortunately too often seen the eye lost after iridectomy—"it did indeed become smaller, but from phthisis bulbi."

Fuchs emphasizes the dangers of iridectomy but adds that a series of favorable results have been obtained. Among the surgeons so reporting are Angelucci, Swanzy and Bergmeister. Haab states that if posterior sclerotomies are begun early enough infantile glaucoma can be cured. Stölting also advises this operation repeated so long as tension remains increased.

Schmidt-Rimpler sums up his study of the literature and the results of his own experience advising that, in advanced cases and in bilateral cases, only one eye be operated on and the result observed, in the meanwhile using miotics in the fellow eye. He is definitely of the opinion that these drugs at least delay the progress of the process.

EXPERIENCE OF THE PROFESSION WITH OPERATIVE PROCEDURES

The following questions were sent to a large number of ophthalmic surgeons throughout the country:

1. What operative procedures have you tried? 2. What have been the results? 3. Do you now favor operative treatment? 4. If so, what is your operation of choice? 5. What per cent. of cases showed evidences of inherited syphilis? 6. What was the percentage of cases in negroes?

About two-thirds of those addressed replied. Quite a number of surgeons either had no clinical or no operative experience with the disease and the experience of no one man was large. The replies to question 5 were disappointing but not entirely unexpected, as in but few cases has the opportunity to employ the Wassermann test been afforded and the answers have therefore been based on clinical signs and family history. Apparently the negro race is not especially susceptible to the affection. If

V. 6
1913

syphilis is a factor in its causation the affection would probably have been seen more frequently in this race.

The replies obtained to the preceding questions are arranged seriatim:

ROY: 1. Repeated paracentesis of the anterior chamber with iridectomy. 2. Very poor. 3. Yes. 4. Same as 1. 5. Almost 10 per cent. 6. Majority in negroes.

FOX: 1. Iridectomy and sclerotomy. 2. Failure. 3. Yes. 4. Enucleation and Mules' operation. 5. None. 6. Low.

WOOD, C.: 1. Iridectomy (ten eyes). 2. Encouraging. 3. Yes. 4. Sclerectomy (trephine), large opening. 5. Twenty-five per cent. 6. Ten per cent.

DE SCHWEINITZ: 1. Repeated sclerotomy. 2. Unsatisfactory. 3. Miotics failing. 4. Either repeated posterior sclerotomies or trephining. 5. ? 6. None.

MAXSON: 1. Galvanocautery to apex of cornea. 2. ? 3. ? 4. ? 5. None.

WESCOTT: 1. None. 2. ? 3. Yes. 4. Iridectomy. 5. ? 6. None.

CHANDLER: 1. Paracentesis. 2. Poor. 3. No. 4. —. 5. ? 6. None.

WILDER: 1. Iridectomy, anterior sclerotomy, excision of the superior cervical sympathetic ganglion, trephining sclerectomy. 2. Immediate results fair. 3. No benefit in the ganglion extirpations. Ultimate results judged to be not good in the majority of cases. 4. Sclerectomy or trephining. 5. ? 6. None.

VERHOEFF: 1. Lagrange, sclerotomy. 2. Excellent in the immediate results. In two cases with advanced changes in which sclerotomy could not be performed, sclerectomy was followed by intro-ocular hemorrhages. 3. Yes. 4. Sclerotomy. 5. ? 6. None.

BRUNS: 1. Iridectomy as for glaucoma. 2. Good. 3. Yes. 4. If seen early glaucoma iridectomy in one eye; treat other eye with miotics. 5. One suspicious. 6. ?

MAY: 1. Iridectomy, three cases. 2. Beneficial for tension and comfort. Vision poor to start with. 3. In suitable cases. 4. Iridectomy. 5. ? 6. None.

WOODS: 1. Iridectomy and enucleation. 2. Two cases with iridectomy; progress arrested. 3. No, unless pain and increased tension present. 4. Iridectomy or similar operation. 5. ? 6. ?

WEEKS: 1. Iridectomy and trephining, after Elliott. 2. Iridectomy not encouraging: trephining sufficient to warrant further trial. 3. Yes, in favorable cases. 4. Trephining. 5. ? 6. Twenty per cent. (one case good result—two years with 1 per cent. pilocarpin locally).

WEBSTER: 1. Iridectomy. 2. Failure. 3. ? 4. ? 5. ? 6. None.

KNAPP: 1. Cyclodialysis; Lagrange without iridectomy. 2. No beneficial results. 3. Where cornea is dull, in lesser grades, pilocarpin. 4. Trephining. 5. None. 6. None.

CALLAN: 1. Iridectomy. 2. Unsatisfactory. 3. Yes. 4. Sclerectomy—punch or scissors. 5.? 6. None.

HARLAN: 1. Iridectomy and opticociliary neurectomy. 2. Poor. Usually alternative enucleation. 3. In some cases. 4. Opticociliary neurectomy. 5.? 6. ?

MARPLE: 1. Iridectomy. 2. Not satisfactory. 3. Yes, in suitable cases. 4. Trephining. 5. None. 6. None.

THOMSON, E.: 1. Iridectomy, sclerotomy. 2. Poor (cases all advanced). 3. Yes. 4. Trephining. 5. None. 6. None.

HANSELL: Never operated for cure.

VEASEY: 1. Only iridectomy. 2. Very few cases. Progress checked in one, but eye enucleated. 3. Yes. 4. Lagrange or Fergus-Elliott operation. 5. None. 6. None.

RISLEY: 1. Broad peripheral iridectomy. 2. With two exceptions, without benefit. 3. Yes. 4. Iridectomy. 5. In one case. 6. One case.

REBER: 1. None. 2. With miotics, results poor, in four cases (six eyes). 3. Yes. 4. Elliott's operation happily adapted to this class of cases. 5. None. 6. None.

GIFFORD: 1. Iridectomy and De Wecker's sclerotomy. 2. Bad with iridectomy; good in one with posterior sclerotomy repeated a second time. 3. Yes. 4. Sclerotomy; trephining. 5. None. 6. None.

KEY: 1. Lagrange with large iridectomy. 2. One case bilateral two years after operation T. + 20-30 mm. of mercury; one eye vision = 20/30, other light perception. 3. Positively. 4. Lagrange. 5. One. 6. ?

SWING: 1. Iridectomy. 2. One case cited eye operated did well, other eye not operated lost. 3. Yes, in progressive cases. 4. Filtering scar operation. 5.? 6. None.

CHATHAM: 1. Ablation of the cornea with subsequent iridectomy in bilateral cases. 2. Poor. 3. No. 5. ?. 6. None.

HOWE: Recalls one case in which he removed the lens, result, finger counting.

WOODRUFF, H. W.: 1. Iridectomy. 2. Bad. 3. No. 5. None. 6. None.

SATTLER: 1. Paracentesis of the anterior chamber. Iridectomy and anterior and posterior sclerotomy. 2. Unsatisfactory. 3. Only in certain cases. 4. Iridectomy. 5.? 6. ?

KOLLOCK: 1. Removed lens in one case. 2. Unsatisfactory. 3. In some cases. 4. Posterior sclerotomy repeated. 5. ? 6. Majority negroes and mulattoes.

HOLLOWAY: 1. None. 3. Attitude against operation. 5. None. 6. None.

BROWN, S. H.: 1. None. 2. None. 3. No. 4. None. 5. One hundred per cent. (in a recent case the Wassermann reaction was positive).

An analysis of the preceding replies shows that iridectomy gave fair results to 42 per cent., and poor results to 58 per cent. of the operators employing it. Sclerotomy gave fair results to 28 per cent. and poor results to 72 per cent. of the operators using it. Paracentesis of the anterior chamber with iridectomy gave unsatisfactory results to 100 per cent. of the operators using it. Sclerectomy gave satisfactory results to 40 per cent., encouraging to 20 per cent., and unsatisfactory to 40 per cent. of the operators employing it. Cyclodialysis gave unsatisfactory results to 100 per cent. of the operators using it. Miotic treatment was on the whole found to be unsatisfactory.

While the number of cases seen by the individual operator was small and the total for any particular operative procedure not large, a summary shows tha. the only method which gave satisfactory or encouraging results to the majority of surgeons employing it, was some form of sclerectomy.

Iridectomy and sclerotomy are the operations which have been longest employed, and it cannot be said that they have inspired confidence except when performed in the very earliest stage of the disease.

The dangers of iridectomy are patent. The sudden release of the intra-ocular tension in an eye in which the natural barrier between the vitreous and the wound has been weakened by disease, is always fraught with danger and often results disastrously. If the disease is to be arrested, the early age at which it is necessary to operate requires the use of a general anesthetic. Vomiting and the natural restlessness of the child after the use of a general anesthetic further increase the danger.

The objections to sclerotomy and to paracentesis of the cornea are that it must be often repeated and even then is often only preliminary to some other form of operation. Of the different ways of performing sclerectomy that of Fergus-Elliott is attended by the least hazard and is easiest of performance. It is inferior to the Lagrange operation in that the filtration area is not so extensive and the scar is not so well placed for permanent effect. The position in which the incision is made in the latter operation is, however, more dangerous. An operation which has been advised by several operators and performed by a few, and which seems to be well adapted to the conditions present in hydrophthalmos, is de Vincentiis. The deep anterior chamber present in these

eyes renders the operation less difficult of performance than in primary glaucoma for which it was devised. That the progress of the disease is sometimes naturally arrested must be taken into consideration in weighing the evidence presented favorable to any operative procedure. Limitation of the progress of the process is not however constant enough, nor does it as a rule occur early enough to justify tentative treatment.

The history in brief of the case of hydrophthalmos with congenital coloboma of the iris from which the specimen for histologic study was obtained was as follows:

CASE 1.—E. B., colored, aged 3 years. Right eye large at birth and has increased but little. Left eye gradually growing larger for past three months. Right eye, broad congenital coloboma of the iris below extreme distention of the globe. Corneal diameter 18 mm. T. + 1 ? (Schiötz tonometer 33 mm. of mercury). Vertical width of palpebral fissure 31 mm. Apex of cornea 15 mm. in advance of its fellow. No view of fundus on account of the corneal haze. Right eye, broad coloboma of iris below. Globe not much enlarged. Diameter of cornea 9.5 mm. T. + ?. Vertical width of palpebral fissure 18 mm.

After enucleation left eye measured 37 mm. in its anteroposterior diameter.

Anatomic Study.—This was made by Dr. Nelson M. Brinkerhoff of the pathologic laboratory of Wills' Hospital, who has furnished the following report: "The globe measured in its anteroposterior diameter 35 mm. and in its longest transverse diameter 28 mm. The anterior chamber was very large; 5 mm. in depth and contained a watery fluid. The crystalline lens was cataractous and dislocated into the anterior chamber. The iris was atrophic and presented a congenital coloboma below measuring 12 by 5 mm. The ciliary body was markedly atrophic. The chorioid and retina were also atrophic to a less degree. The sclera was thinned and stretched throughout. The nerve was atrophic and deeply cupped. The vitreous chamber was filled with a brownish-yellow fluid.

"*Microscopic Examination.*—Cornea was thin. The layers were all present; there being some separation of the middle third of the substantia propria. The iris tissues were atrophied. The chromatophores were decreased and the pigment epithelium increased. Schlemm's canal was absent. The angle of the anterior chamber was completely obliterated by connective tissue which, with some endothelial elements structurally arranged, probably represented the pectinate ligament. The ciliary body was atrophic. The processes were stretched, elongated and superimposed. The retina was degenerated, atrophic and cystic. The epithelium however was increased. The nerve-fiber layer was thickened and the molecular layers in several positions had coalesced. The rods and cones were well developed. The chorioid was atrophic, there being excessive proliferation of the connective tissue throughout which almost obliterated it; it being represented for the most part by an opaque pigmented line. There was general thinning of the sclera. The nerve was deeply cupped, and formed mostly of connective tissue. Some of the cells were pigmented. The subdural space was obliterated and the subarachnoid partially so." The conditions present place the process in the first of Magitot's classes. The condition of the angle of the anterior chamber suggests the descriptions given of it by Collins.

Dr. E. V. L. Brown, who kindly looked over several of the specimens, finds an absence of Schlemm's canal, aplasia of the entire venous system of the angle and obliterating endophlebitis of the scleral emissaries from the canal area and of some of the anterior ciliary veins; and on the side of the coloboma also of some of the anterior ciliary arteries. There is incomplete separation of the iris from the cornea. There is no prenatal iris tissue, over the corneoscleral trabeculae. The spaces of the trabeculae are wide open and unobliterated, though the individual trabeculae are rich in endothelial nuclei. He believes that the absence of Schlemm's canals and venous system better explains the production of hydrophthalmos in this case than the incomplete separation or persistence of prenatal pectinate ligament (Collins), as in the sections studied by him the anterior chamber is not sufficiently cut off from the trabecula spaces to bring on hydrophthalmos.

CASE 2.—In the fall of 1910 I had referred to me, at St. Agnes Hospital, A. B., a white child, aged 3 years, with bilateral hydrophthalmos. The globes were very large and the tension moderately high. Vision was evidently much affected as there was nystagmus. The child, though well nourished and healthy, was very fretful. Photophobia was extreme. Under ether anesthesia I attempted iridectomy, making the incision at the limbus. On completion of the section the lens and considerable of the vitreous extruded. The eye was lost from the resulting iridocyclitis. In the following spring, not having accommodations for the child I referred it to Dr. Posey at the Children's Hospital. Dr. Posey informs me that he first attempted cyclodialysis but failed. Subsequently he did a sclerotomy and later an iridectomy but the progress of the disease was unchecked.

I am indebted to Dr. C. A. Veasey of Spokane for the following case history and also for the eye which it was found necessary to remove. The anatomic study was made by Dr. Nelson M. Brinkerhoff who has kindly written the report of the findings:

"The patient, a boy, was first seen by me Oct. 7, 1908, at the age of 15 months. No family history of any unusual condition on either side could be elicited. At birth the right eye was found to be very much larger than the left and it had been gradually increasing in size up to the time of my examination, when the child was 15 months old. The tension was decidedly increased. The cornea was so hazy and the patient so young that it was impossible to ascertain the condition of the media by ophthalmoscopic examination. I do not know whether the lens was opaque or the optic nerve was cupped. On Oct. 12, 1908, I performed an iridectomy under ether anesthesia. Healing was uncomplicated, and the patient was seen several times between this date and June 19, 1911. During this period the increase in the diameters of the eyeball seemed to have been entirely checked. At this time, and for several weeks before, the parents stated that the child had displayed such great photophobia in the left eye that it was impossible to get him to stay out of doors in the bright light, and whenever he was exposed to the light he attempted to shield his eyes, although there was absolutely no macroscopic indication of any irritation whatever, externally or internally in either eye. This condition had continued for some time and had so alarmed the parents that on June 19, 1911, I enucleated the buphthalmic eye under ether anesthesia. The patient has been seen twice and apparently the photophobia of the left eye immediately cleared up and he has worn a glass without any difficulty. The left eye has been normal in all respects."

Anatomic Study.—"The globe measured in its sagittal diameter 32 mm., and in its vertical diameter 29 mm. The cornea was clear. There was a staphyloma of the sclera immediately behind the corneoscleral limbus. The anterior chamber measured 5 mm. in depth and contained no fluid. The lens was cataractous. The iris and ciliary body were thickened; the former presented an artificial coloboma. The vitreous chamber was filled with clotted blood. The retina was detached except at the ora serrata and nerve-head and was umbrella-shaped, occupying the anterior and central portions of the chamber. There was no apparent change in the chorioid and no appreciable cupping of the disk.

Microscopic Examination.—"The epithelial layers of the cornea were somewhat thickened. The superficial cell layers showed cloudy swelling. Descemet's membrane was normal, except for a single rupture, which may have been an artefact. On the posterior surface of the membrane there was a fine homogeneous deposit. The anterior chamber was obliterated, being filled with a similar exudate and with blood-cells. The iris was atrophic and the stroma elements were almost completely degenerated; at the pillars of the coloboma the pupillary border was curled on itself anteriorly. The ciliary bodies were atrophic and cystic. The remaining stroma was almost entirely composed of pigmented connective-tissue cells. The

V. 6
1913

ciliary muscle was almost completely atrophic and elongated by traction. The pectinate ligament was obscured. Schlemm's canal was absent except at one point where it was represented by endothelial cells. The chorioid was atrophic and there was a proliferation of its connective tissue elements. The blood-vessels were obliterated. The vitreous chamber was filled with a fine granular exudate and free blood. The retina was degenerated. There were traces of its nuclear layer, and excessive pigmentation in the posterior zone. The sclera was thinned, the anterior portion more so than the posterior. The optic nerve was atrophic and the disk slightly cupped."

The study of these two eyes lends additional support to the view that the essential factor in the production of hydrophthalmos is an absence or incomplete development of the canal of Schlemm and that a probable contributing factor is the presence in the angle of the anterior chamber of prenatal connective tissue.

1819 Spruce Street.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. SATTLER, SCHOENBERG AND ZENTMAYER

DR. LEWIS H. TAYLOR, Wilkes-Barre: We are still far from the solution of the problem of glaucoma. Dr. Sattler raises the question as to whether increased tension be the first and actual dynamic factor in glaucoma. Whether it be or not, it is generally true that it presents itself as the chief factor in the cases when they are brought to us. At least this has been my experience and I think it has been yours. There are others but we do not usually have the chance to see them as the patients do not present themselves to us until the disease is well advanced. Dr. Sattler asks many questions which we would all gladly answer if we could. As I understand it, his view is that acute glaucoma and subacute or chronic forms are different processes and from different causes and this may open to us a new thought worthy of consideration. Modern viewpoints of glaucoma will include of course modern operations. Dr. Weeks, in a full discussion of this subject a few months ago before the Pennsylvania State Medical Society, described nineteen different operations with several modifications of some of them. I think we shall all agree that at present the operation that seems to meet with most favor is that of trephining the sclera. I have seen the operation of Ellictt and some others performed, but thus far I have only performed the operation of iridectomy—the operation that Dr. Risley holds to be, if properly performed, the best of all—and so I am not prepared to speak from personal experience but only from observation of the newer method. I agree with the author that it matters little which operation is performed if the operator thereby can do a little good, stay the final extinction of function and at the same time avoid doing harm. With some ophthalmologists it is also a modern conception of glaucoma that all operations are of doubtful value, especially in hemorrhagic glaucoma, except that of enucleation. I agree with a writer of large experience as an operator who has recently said that it was a rule with him never to operate for glaucoma if the tension could be held at normal and loss of field and vision prevented by miotics.

DR. EMMET L. JONES, Cumberland: The experience I have had with glaucoma is not large but is in a direction entirely different from that in which everybody else has been looking. I have believed for a long time that glaucoma is not primarily a disease, but that it is a symptom of no man knows what, so far as I have been able to learn, and we may find a cure before we know what we are curing. I have had more experience in treating glaucoma complicating traumatic conditions than in primary glaucoma, but I have had some experience with acute, sudden, fulminant glaucoma, chronic glaucoma and in cases long totally blind and so intensely painful that they were usually removed. The plan of treatment has been sub-conjunctival injections. I do not know what brings about the cure, but the results have been very satisfactory. Whether it is due to circulatory or lymph stasis, whether it is due to hardness, or whether the hardness is simply one of the symptoms I don't know. With the injections of cyanid I do not use dionin but have used epinephrin (adrenalin), and a single injection is usually all that is necessary. In Barrier's later book he speaks of using 1 to 1,000 iodate of soda. I have used that in some cases and the results are certainly marvelous in some instances. I do not know whether it removes the stasis or whether the continued pressure causes the emptying of the eyeball, but the lid swells up and the pressure continues for several days. One patient was a prominent attorney in the sixties who was seized with a fulminant attack of glaucoma in the night and did not know that he had lost his sight until the next day. Under profuse purging by salines, and miotics, eserin, etc., in twenty-four hours the violent pain had left but the eye was totally blind. I gave the injection. It is horribly painful, but in thirty minutes the pain had ceased and the patient went back to his office. He never had any pain afterward. In the course of two or three days vision was restored, the swelling remained some days, there was some transient diplopia, but the eye was absolutely restored without a vestige of after-effect. In some cases of these old totally blind glaucomas, I have seen the pain stop and the eyeball stay hard as wood. Likewise in chronic glaucoma it seems to be serving well, as far as I have been able to use it. It does better than other measures. The strength is from 1 to 1,500 of the cyanid and 1 to 1,000 of the iodate, using thirty drops. The solution soon decomposes into iodin and something else and must be made fresh.

DR. ADOLPH ALT, St. Louis: After Dr. Schoenberg's first paper had appeared in the March, 1913, number of Knapp's *Archives* I have repeatedly taken occasion to verify his statement that leaving the tonometer for a certain number of seconds on the cornea will reduce the intra-ocular pressure. Dr. Schoenberg attributes it to the influence which the weight of the tonometer exerts on the ocular drainage. This drainage, which in a normal eye, goes on continuously, takes place by means of preformed channels. The generally accepted opinion is that the drainage takes place solely into the venous circulation. I should not recall this to you, had not Lebouecq in the *Bulletin de la Société Belge d'Ophthalmologie* of April, 1913, published a series of experiments, by the results of which he considers it proven that the drainage of the anterior and posterior chambers takes place by means of the lymph-vessels accompanying the veins and is distinctly separated from the drainage of the posterior parts of the eyes. Thus, whether these findings prove to be correct or not, our seemingly well-established knowledge about the manner of ocular drainage is suddenly assailed again. That the ocular drainage must depend on the patency of these innominate channels seems indisputable. It naturally follows that anything interfering with this normal ocular drainage must lead to an increase in the intra-ocular pressure, and it is rather astonishing that Dr. Schoenberg found that an iritis in rabbits did not seem to influence the drainage. It seems to me that the experiments which led to this conclusion are the least conclusive in his series. Although his experiments regarding the action of the extrinsic muscles on the intra-ocular pressure cannot be called altogether conclusive, it seems impossible to conceive that the action of these muscles should not exert any influence on the intra-ocular pressure. Dr. Schoenberg's experiments referring to the relation of the drainage to the intra-ocular pressure, and especially to eyes which are suspected of a tendency to glaucoma, are of decided practical value. The fact which he has elicited that a reduced rate of drainage may mean a latent glaucoma, although the intra-ocular pressure may be below 26 mm. Hg, seems to be especially so. That the increase of the intra-ocular pressure in glaucoma is not disease and that even so-called primary glaucoma must be looked on as a symptom consequent on some as yet hidden affection, is, I do not doubt, the general opinion. Yet, while treating symptoms is not the most scientific way of managing disease, it may be justified in glaucoma, until more is known about it. It is as rational to interfere surgically in order to relieve the pressure and its dire conse-

quences as are many similar measures in general surgery. Of course, when the primary cause is once known this must be taken care of, and yet, even then, it may still be, perhaps, a very rational procedure to establish quickly a reduction of the intra-ocular pressure.

DR. L. WEBSTER FOX, Philadelphia: Even in the large clinics of the East, where we see a great many cases, no ophthalmic surgeon has seen many cases of buphthalmos. I have seen probably five or six cases in my experience, and have found that it is a congenital defect, prenatal in its origin, in which usually at about the third month of intra-uterine life, there is a lack of development in the anterior part of the eye; the canal of Schlemm is absent and the result is an overdevelopment of the organ due to intra-ocular pressure and stretching of the tissues, whereby they are enlarged to an inordinate degree, producing buphthalmos, dropsy of the eyeball or the so-called "eye of Juno." I have tried various methods of treatment, including iridectomy and have felt that anterior and posterior sclerectomy have given me the best results temporarily, but they have ultimately failed and the eye has shrunken. I recently operated in a case according to the method employed by Dr. Spratt in the case he exhibited. I believe that this, which is the Elliott operation, is the one most suitable in this dread disease. My experience in the last two years with the Elliott operation for glaucoma leads me to this belief. Dr. Batroff and I, in a paper read before the Colorado Medical Society a few years ago, stated that in many of these cases of glaucoma an interstitial nephritis and an increase of blood-pressure was found. A few years ago, when Mr. Treacher Collins visited my clinic in this country, we had a case of acute glaucoma, with high blood-pressure, and also a high degree of intra-ocular tension. This patient had nearly 100 mm. pressure with the Schiötz tonometer. The eye was as hard as stone and vision was gone. This was a second attack; the patient had lost the other eye in an operation by a clever operator, who attempted an iridectomy and had what any surgeon might have had, a gush of vitreous with hemorrhage and destruction of the eyeball, necessitating enucleation. Taking this experience as a warning, I tried first of all to reduce the blood-pressure. The patient was bled, the sphygmomanometer applied, and the pressure was reduced from 275 to 150. Eserin was applied to the eye every half-hour for twenty-four hours, with no miotic effect until the blood-pressure was reduced, when we immediately obtained contraction of the pupil. After that I was able to perform an iridectomy with a satisfactory result. If the operation of Elliott does as much good in all cases of buphthalmos as in the little child we have seen here to-day, great advances will have been made in the treatment of the disease. I feel that LaGrange has accomplished the same thing in his operation as Elliott has with the trephine, but the man who has few operations cannot do it so well as one with a great deal of experience. I believe the Elliott operation is the one for the majority of operating surgeons.

DR. JOHN O. MCREYNOLDS, Dallas: I wish to add my endorsement to the method of trephining advocated by Colonel Elliott of Madras. Two years ago, at the Oxford Ophthalmological Congress, I had the pleasure of learning this method directly from Colonel Elliott, and my conviction as to its high value has grown stronger in the intervening time. Colonel Elliott's method of making the flap seems to have practically attained perfection. There may be, however, some room for study and discussion as to the best size and pattern of trephine to be employed, and the indications are that a large variety of such instruments will be employed by different men to accomplish the same result. But this is merely a matter of detail and only emphasizes the cardinal virtues of sclerectomy, a procedure which has been so thoroughly developed by our colleague of Madras. I have been very much pleased with a substitute for trephining itself, by the employment of a small scleral knife and a small scleral stitch in the following way: 1. Prepare the flap essentially according to Elliott. 2. Take a fine, sharp full-curved needle carrying black silk thread. 3. Introduce this needle into and through the sclera 2 mm. from the cornea, carrying the point forward to the anterior chamber from the angle of which the needle should now immediately emerge through the periphery of the cornea. 4. This will enable you to grasp securely 2 mm. of sclera in the loop of the thread which can now be held gently taut with the left hand. 5. Take a small scleral knife (a Beers knife will do) and by gentle strokes mark out a semicircular incision just enclosing on the outer side the small portion of the sclera in the grasp of the thread. 6. As the incision passes through the thickness of the sclera, the flap thus formed will be drawn by the thread towards the cornea, and the scleral flap may be thus extended by the knife until the anterior chamber is opened by its periphery, when the sclerocorneal flap may be excised as far forward as desired, taking care not to button-hole the reflected conjunctiva. 7. A portion of the iris should be excised leaving, however, the sphincter of the iris untouched, thus producing a button-hole in the iris which is quite sufficient and can be seen through the transparent conjunctiva when replaced. 8. The preservation of the sphincter means diminished trauma, better protection for the lens, increased efficiency of miotics, if subsequently employed, and a better looking eye. I have employed these procedures in both chronic and acute glaucoma with the most gratifying results.

DR. ALLEN GREENWOOD, Boston: I cannot help but feel that acute and chronic glaucoma are practically manifestations of one and the same disease. I think that every one of you has had the experience of a simple chronic glaucoma changing to the acute form under some circumstances of mental strain. I believe the trephine operation is one that has come to stay, but having commenced five years ago with the LaGrange operation I have continued it. During the five years, I have operated on twenty-five patients. Three years ago at the request of some of the gentlemen from St. Louis I operated on a patient before this Section at the hospital there, and I have just been informed that the patient still retains the vision restored after the operation. In the twenty-five LaGrange operations of five years' or less standing, some perhaps within two or three months, I have seen one eye lost. The majority of my patients on whom I performed the LaGrange operation have to-day a better field of vision than they had before the operation was performed. It seems to me that this is one of the crucial tests of the advantage of a LaGrange or a trephining operation. It is performed to obtain a filtering cicatrix and most of these patients to-day show a sort of boggy condition of the conjunctiva just above the limbus. It looks honeycombed, and we know that these patients are seeing and using their eyes in almost a normal manner. With regard to the use of miotics: in any case of chronic glaucoma where the field of vision and the vision itself can be retained as it is, and the patient has previously lost one eye, I very seldom operate, but where the vision is going down, as in the case of the patient in St. Louis, who had lost sight in one eye, I perform a LaGrange operation.

DR. WALTER R. PARKER, Detroit: I have always been interested in the fact that glaucoma may possibly be due to an obstruction of anterior or posterior filtration channels as mentioned by Dr. Alt. We have all seen cases of glaucoma in which our irridectomies have entirely failed. It seems to me there is a possibility of glaucoma being a posterior filtration disturbance, which is manifested by a shallow anterior chamber which would not be helped in any way by an irridectomy. On the other hand, a glaucoma that is dependent on obstruction of the anterior filtration channels would have some chamber left, in fact, it might be nearly normal; in these cases, I find iridectomy does better. In determining whether an iridectomy will benefit or not, the depth of the anterior chamber is important. The deeper the chamber the better are the chances of benefit from an operation. I have had experience with the Elliott operation in twenty-three cases. All but three had the tension permanently diminished. In each of the three failures there were some inflammatory symptoms present. While the Elliott operation is becoming the operation of choice I think it should be limited to the non-inflammatory cases. In two cases of buphthalmos, I have performed a double iridectomy. In one no effect was noted, but in the

second the tension was reduced from 70 and 74 to 30 and 34, respectively. Whether there will be a permanent benefit I am unable to say. In both of these cases the Wassermann was negative.

DR. S. D. RISLEY, Philadelphia: I should like to have Dr. Sattler tell us in his closing discussion whether there is any clinical method by which the probability of the claims of Fischer as to the existing acidosis of the tissues may be demonstrated.

DR. LUCIEN HOWE, Buffalo: For those who are interested in the subject it may be worth while to mention that these cases of buphthalmos and anophthalmos are treated with wonderful clearness and exactness in Eversbusch's "Augenerkrankungen im Kindesalter." A translation into English of this recent work is now being made.

DR. G. C. SAVAGE, Nashville: If it is true that Dr. Fischer is receding from his original teaching I regret it very much. I want to use this fact as an illustration of receding from a truth: Brown-Séquard, as everybody knows, was a strong advocate of the crossing of the nerve fibers from one side to the other. I heard him try to destroy that theory, yet it was a truth that he was trying to destroy. What we have heard here is a demonstration that here is a condition of the tissues of the eye which predisposes to glaucoma. Let us fight our way into the daylight concerning glaucoma, and let me beg of you, Dr. Sattler, to call Fischer back to his original teaching, even if he should find that a condition of the blood causes the acidosis of the tissues of the eye. I have never injected sodium iodate, but often have injected sodium citrate. I turn trembling from that to operative procedure, sometimes the trephine and sometimes iridectomy. In almost every case in which I have turned I have regretted it. But if in addition to the injections we can put something into the circulation that will help to cure the cases, let us do it. I am now treating a case of hemorrhagic glaucoma, and I venture to say that Dr. Risley would not operate in such a case for any consideration whatsoever. I treated the patient with eserin until she was losing her vision, and gave the poor woman, who was dependent on her daily labor for her subsistence, injections of sodium citrate in each eye once a week. Vision is almost normal in one eye and rapidly gaining in the other and she is at work every day, even on the days of the injection. Dr. Alt, I thank you from the bottom of my heart for the suggestion of the internal administration of calcium chlorid. The local condition of the eye leading to glaucoma may be superinduced by a state of the blood, which calcium chlorid may destroy.

DR. JESSE S. WYLER, Cincinnati: I should like to make a few remarks on Dr. Fischer's theory. Dr. Reubens of Heidelberg had studied Fischer's theory carefully and determined to work out three questions. The first was, what part of the enucleated eye takes part in the colloid swelling. We all know that the enucleated eye does swell. He found by producing this swelling in the eyes of rabbits and human eyes and then examining them, that the cornea and the sclera alone partook in the edema. This question then arose: How is the tension raised when the cornea and sclera alone are edematous? He found that the size of the ball did not increase externally but that the swelling took place inwardly, so that the sclera was increased in thickness internally and thus the area of the interior of the eyeball was decreased; the exterior remaining the same, and the contents remaining the same, the tension was raised. The third question was, Does this theory work out in living eyes the same as in enucleated eyes? He took a series of rabbits and injected subconjunctivally from 1 to a 10 per cent. solution of hydrochloric acid, which produced a great increase in tension, in some as high as 170 mm. He then reduced this tension by giving subconjunctival injections of sodium citrate, 5 per cent. Then, proving to his satisfaction that this worked in the laboratory, he determined to try it on human beings. But as so often happens, laboratory and clinical methods do not agree, and Reubens found every time he used the sodium citrate with the tonometer readings that the tension was not reduced. I personally tried citrate of soda in eight glaucoma cases, three acute, three of the chronic and two of the simplex variety. In none was the tension decreased; in fact, it was raised 5 or 10 mm. after the injection. In regard to the influence of blood-pressure, Kraemer von Graefe's *Archiv* published the results of work done in Fuchs' clinic. He selected at random fifty cases of senile cataract and took the blood-pressure with the Riva-Rocci instrument; then he took fifty glaucoma cases and found the average pressure about the same in both conditions. He could not find the slightest basis for the theory that increased blood-pressure had anything to do with the causation of glaucoma.

DR. OSCAR WILKINSON, Washington, D. C.: I think high blood-pressure has a great deal to do with the treatment of this disease. In my cases of chronic glaucoma I invariably try to reduce the blood-pressure and I believe that eserin and miotics have the better effect. I get better vision and an increased field of vision. I first give small doses of calomel and the hyposulphite of soda. Another remedy which I give in this class of cases is 2 or 3 grains of mercuric chlorid in saturated solution of potassium iodid, which gives the biniodid of mercury. The mercury is an antiseptic and the iodid gives an alterative effect. Diet plays an important part in the handling of these cases. I take them off of meats, except chicken or game and fish. Even the buttermilk diet is good. When the diet is controlled and an alterative has been given, the chronic cases of glaucoma are in a measure under control, and I think this has a direct influence on the case—as much as eserin itself. The trephine operation is the ideal one for the ordinary practitioner. I have had experience in several cases, and in one of these in particular, in which there was high tension due to an attenuated cornea, the tension was reduced to normal and the size of the eye was materially reduced.

DR. W. H. WILDER, Chicago: The results of Dr. Schoenberg with the tonometer confirm the belief that in glaucoma we are dealing with a condition in which there is some obstruction of those channels which allow the escape of fluids from the eye rather than in the vessels that admit blood to it. It seems to me that we have a clue to the subject in the condition of hydrophthalmos which we know to be due, in some cases at least, to the obliteration or lack of development of the spaces of Fontana or the canal of Schlemm. It cannot be that everyone is liable to glaucoma, for after all it is comparatively infrequent, and we must realize that only certain individuals are susceptible to it, and these for some reason, may have some peculiar condition of the tissues of the eye, or of the vessels concerned with the outflow of fluids from the eye, that render them liable, with a suitable exciting cause, to an attack of glaucoma. Persons who do not possess this peculiar type of vessels will not develop glaucoma, even if subjected to the causes that would precipitate an attack in those so predisposed. Hence, work like this of Dr. Schoenberg's may enable us to determine by means of the tonometer the degree of patency of the channels of outflow of the eye. All our operative procedures are merely remedial and do not reach the base of the trouble. I was hopeful that the studies of Fischer on this subject would give us something of a working hypothesis, but in the Illinois Eye and Ear Infirmary, where we tried the injections of sodium citrate in the acute, chronic and simple forms of glaucoma, we could not see that the patients derived any material benefit.

DR. EDWARD JACKSON, Denver: We appreciate especially Dr. Schoenberg's observation that retarded drainage may be of real diagnostic value. I think we shall soon come to realize that the absolute height of the intra-ocular tension does not furnish a satisfactory basis to decide the presence or absence of glaucoma. Priestly Smith's definition that glaucoma is increased intra-ocular tension plus its causes and effects, is the best definition. But there is a clinical entity which is something more than increased tension; and increased hardness of the eyeball can exist outside of that clinical condition that we characterize as glaucoma. I have under observation a patient whom I have had the opportunity of watching for about seven years, in whom one eye is entirely blind and

both eyes present typical glaucoma cups. Only once in that time have I been convinced that the tension was above normal, and since I have used Gradle's modification of Schiötz's tonometer I have never found it above normal; but there is not the slightest question that the man has glaucoma. On the other hand, within the last year I have observed three cases, in young women, in whom the intra-ocular tension rose to about 50 mm.—one to about 60 mm.—with the clinical symptoms of an acute, mild attack of glaucoma, some redness, very distinct pain and marked dilatation of the pupil. In two cases, this followed needling for congenital cataract. The other was a case of tuberculous chorioiditis at the macula. All of these eyes have gone back to normal condition without any radical treatment for glaucoma, although when I first found that the tonometer was registering something like 60 mm. of mercury I was afraid they were starting on a course of glaucoma. The height of the intra-ocular tension at the time of one observation is not an indication that can be wholly relied on to decide as to the presence of the disease that we know as glaucoma. Therefore the symptom of diminished intra-ocular drainage may be one of even greater significance.

DR. CHARLES N. SPRATT, Minneapolis: The little girl with the buphthalmos who was exhibited here this morning is 6 years old; the father and mother are second cousins. At the age of 3 or 4 months it was noted that there was something wrong with the eyes, which were sensitive to light. Only within the last two years an increase in the size of the globe has been particularly noted and photophobia has been marked. We took the tension of the left (blind) eye of the patient, the tension being plus 1. Trephining was performed, and was followed by a gush of yellowish aqueous that came out of the top of the trephine. The patient moved during the anesthetic and lost some of the vitreous. She struggled considerably after the operation and the dressings were stained with blood that evening; there was considerable hemorrhage. In the right eye the vision was equal to reading large newspaper print at 4 or 5 inches. We did a paracentesis to reduce the tension. It remained down and forty-eight hours afterward trephining was performed on that eye. The tension has been much less than before the operation; the patient has had less pain, sleeps better, is better-natured and the eyes have not been as red. Regarding vision, it is difficult to say as she still has considerable photophobia. If something is not done in these cases they will result in total blindness. Iridectomy, as has been said, is not a safe procedure, and trephining, with a preliminary paracentesis, is the only legitimate thing to be done. I had a similar experience with an old lady with glaucoma absolute on whom an iridectomy had been performed eighteen years ago. I did a trephining and a severe hemorrhage followed. The eye was lost with iridocyclitis. Reducing the blood-pressure by blood-letting and thus reducing the tension of the eye may be all right, but it seems to be a little in the way of the Chinese method of making a roast pig by burning the house down.

DR. ROBERT SATTLER, Cincinnati: The discussion has brought out varied opinions concerning the nature and treatment of glaucoma in general. I will only add a few words and repeat the main purpose of the paper; that modern opinions of the pertinent facts of glaucoma restrict its scientific interpretation. Formerly every hypertonic globe was so designated. Our present understanding of the origin and pathology of primary glaucoma should confine the name to its two distinctive clinical phases—chronic non-inflammatory and acute or subacute inflammatory glaucoma. Dr. Savage has referred to the Fischer treatment. My reason for not taking this up more fully at present is that this subject was discussed in a special paper read before the Cincinnati Research Society, May, 1913. Now as formerly, we give Fischer's treatment a trial in selected cases, but have given up the local or subconjunctival injections of citrate of soda solutions. We adhere faithfully to Professor Fischer's suggestions concerning the internal administration of alkaline remedies and the use of slow rectal injections of citrate of soda in certain cases.

DR. MARK J. SCHOENBERG, New York: I expected more criticism. Dr. Alt said that my experiments on the reflex action or relation between distant organs and the intra-ocular pressure were not conclusive. Many of my experiments are not conclusive. I came here with only a very incomplete record of certain series of experiments. I only tried to stimulate work along the same line. Dr. Alt says we cannot give up local treatment. I did not say anything about giving it up. I said it is not rational, and it is not. It is like stripping the kidney of its capsule in some forms of nephritis. Nephritis cannot be cured in that way; neither can we cure glaucoma by any kind of an operation on the eyeball. The present tendency to devise operations for the relief of tension is not rational. The essence of glaucoma is not increase in tension, just as high blood-pressure is not of arteriosclerosis.

DR. WILLIAM ZENTMAYER, Philadelphia: The cumulative evidence we now have that infantile glaucoma is due to the faulty development of Schlemm's canal will be of assistance in pointing out the pathogeny of glaucoma. Of course it is well recognized that the disease described in my paper is true glaucoma. The only reason the symptoms are different is the difference in the tissues of the immature and the mature eye. I want to caution against operation in advanced cases of this affection. One of the reasons for writing the paper was the disastrous result in such a case. I tried to do an iridectomy and lost the eye at the time of the operation. The vitreous lens extruded through the wound. The child returned to the city later and I was compelled to turn the case over to a colleague, who tried to do a cyclodialysis and failed. Subsequent operative procedures likewise failed. In regard to Dr. Spratt's remarks I would like to say that the prognosis is not so grave for unoperated eyes as he states. A great many cases are on record in which the process was naturally arrested. This is explained by Treacher Collins on the ground that the trouble is a persistence of the fetal ligamentum pectinatum in the angle of the anterior chamber, and these attachments are broken loose as the eyes grow and a natural cure is effected. The LaGrange operation I believe is in many respects superior to the trephine. The cicatrix is larger, is better placed, so as to open the suprachorioidal space better, but is not so simple as the trephine operation and should not be attempted in this type of glaucoma.

TRACHOMA, ITS PREVALENCE AND CONTROL AMONG IMMIGRANTS

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For the purpose of this paper, trachoma, or granular lids, may be defined as a communicable disease of the conjunctiva of unknown etiology, chronic in character, and prone to remissions and exacerbations. It is characterized by granules or the so-called trachoma bodies, with more or less hypertrophy, is most resistant to treatment and is always followed by the stage of cicatrization. By reason of its communicability, chronicity of course and often disastrous results, trachoma is the most important of all the inflammatory diseases of the conjunctiva.

The dissemination of trachoma has often been charged to Napoleon's soldiers when they returned to Europe from Egypt, where they suffered extensively with a distressing ophthalmia. It is undoubtedly of far more ancient origin, however. It is supposed to have been originally endemic in the Orient, whence it extended gradually to the westward. Trachoma has usually been considered as an exotic disease, but recent investigations have shown that our Indians are heavily infected and that the inhabitants of at least parts of the Appalachian range of mountains suffer from this disease to an alarming extent. Further investigations will more

V. 6
1913

than likely show a surprising amount of this disease. The disease prevails in Turkey, Russia, Southern Italy, Austria-Hungary and Greece. It has been generally believed that the negro is immune to trachoma, but numbers of well-marked cases are annually detected from the West Indies. One of the most marked and stubborn cases that has been treated at Ellis Island was in a negro girl. Ninety per cent. of the population of Egypt are said to be afflicted with trachoma. The wide-spread prevalence of this disease there renders it necessary for the British government to maintain a system of ophthalmic hospitals for the relief of these unfortunate people. The medical examination of all arriving aliens is imposed by law on the medical officers of the United States Public Health Service. Section 2 of the immigration law as amended in 1910 provides for the exclusion from admission to the United States of the following classes of aliens:

Idiots, imbeciles, feeble-minded persons, epileptics, insane persons, all persons who have been insane within five years, persons who have had two or more attacks of insanity at any time previously; paupers, persons likely to become public charges, professional beggars; persons afflicted with tuberculosis or with a loathsome or dangerous contagious disease; persons not comprehended within any of the foregoing excluded classes who are found to be and are certified by the examining surgeon as mentally or physically defective, such mental or physical defect being of a nature which may affect the ability of such alien to earn a living.

The "Book of Instructions for the Medical Inspection of Aliens," revised Jan. 18, 1910, classifies trachoma as a "dangerous contagious disease," which puts it under the provision of the immigration law and makes mandatory its deportation.

Section 9 of the immigration law provides that a fine of one hundred dollars be imposed on the steamship company for every "dangerous contagious disease" brought to the United States which might have been detected by means of a competent medical examination at the time of foreign embarkation.

Many prominent ophthalmologists of this country had insisted that trachoma was a communicable disease which caused disastrous results to the eye and should, therefore, not be permitted to enter this country. Trachoma was classified as a dangerous contagious disease in 1897 and the credit is due to some of the leading ophthalmologists of this country. Prior to this time it was not so classified and undoubtedly many aliens were landed here suffering with this disease, thus establishing foci of infection.

Trachoma thus became an excludable disease and, since 1897, thousands of persons with it have been excluded from landing. Fewer cases of trachoma are now found among immigrants and only a very few of the old and worst cases, "Egyptian ophthalmia," with its distressing complications, as compared to the numbers formerly encountered. This must be attributed to the fact that the steamship companies maintain an inspection service abroad in an effort to prevent the embarkation of persons of this class, which would mean deportation and possibly fines, especially the latter. A few hundred dollars in fines have a more deterrent effect than many dozen deportations. It has been stated that the steamship companies are rejecting in Europe on account of trachoma thousands of applicants for passage each year. This means that there is an accumulation of these persons ready to take immediate advantage at any time the laws and regulations might be less stringent in this respect. The careful examination made of all eyes on arrival not only prevents trachoma from being brought here, but deters those suffering from this disease from embarking. In spite of this, there were twenty-five hundred aliens found to be suffering from trachoma and so certified during the fiscal year ended June 30, 1911.

Immigration to our shores during the past ten years has been nearly one million annually. In 1907 it reached its highest point, when 1,300,000 were admitted. During the past fiscal year about 70 per cent. of the total immigration came from Eastern and Southern Europe and Western Asia. Nearly 40 per cent. of the total for that year came from two countries, viz.: Russia and Italy, the former mostly from Southern Russia and the latter from Southern Italy. About 11 per cent. came from the various provinces of Austria-Hungary, 3 per cent. from Greece and 2 per cent. from Turkey (Europe and Asia).

At the port of New York (Ellis Island), which is the gateway for about 75 per cent. of our immigration, 750,000 aliens were examined during the fiscal year ended June 30, 1911, and 1,167 of them were found to be suffering from trachoma. The past fiscal year 718 cases of trachoma were detected among 725,000 arriving aliens. During the six years from 1907-1912 there were 6,657 cases of trachoma certified at Ellis Island—an average of 1,100 cases annually. Of the 1,167 cases certified at Ellis Island in the year 1911, nearly 60 per cent. came from Russia and Italy (South), both countries furnishing about the same amount; about 16 per cent. from Austria-Hungary, about 13 per cent. from Turkey and the remainder from Greece and various other countries. Of the 718 cases certified at Ellis Island during the last fiscal year, Russia furnished 35 per cent.; Austria-Hungary, 22 per cent.; Italy, 14 per cent.; Turkey, 17 per cent., and Greece less than 5 per cent. It will thus be seen that the majority of trachoma among immigrants comes from Russia, Italy, Austria-Hungary, Turkey (Armenia, Syria, etc.) and Greece. Fortunately we get but little immigration from Egypt, where the disease is so distressingly prevalent.

Statistics show that this disease is found most extensively among the following races: Syrian, Armenian, Italian (South), Hebrew, Polish, Greek, Lithuanian and Slovak.

While the national government does not attempt to pass on the mental and physical condition of the immigrant until he arrives at one of the ports of entry of the United States, the steamship companies have instituted a system of medical examinations of all emigrants prior to embarkation. I am informed by a representative of one of the steamship companies that the following method of examinations is now practiced abroad: The steamship companies have established near the German border what they term "control stations," or detention houses for emigrants originating in Eastern Europe, Russia, Austria-Hungary, etc. Emigrants are held at these detention houses or "control stations" until a sufficient number have accumulated, when they are forwarded to Bremen, or wherever it is the intention to embark them. There are about ten of these "control stations." Before the emigrants are permitted to leave the "control station" they are examined as to their physical and mental condition by a physician who is employed especially for this purpose, and those passed proceed to the port of embarkation. Those in whom some diseased condition of the eye is found of sufficient importance are held at the control station for treatment, if it appears that the condition might be easily curable. If a case

is recognized as trachoma, however, and prolonged treatment required, the patient is sent home and advised to try again after having his eyes treated and cured. On arrival at the port of embarkation they are put in the company's detention house and are examined again prior to embarkation by the ship surgeon and a local physician in the employ of the company, with a view to another weeding-out process. During the voyage the doctor is supposed to make a daily examination and isolate such eyes as might convey contagion.

Trachoma without secretion is probably but slightly infectious, but the onset of an acute ophthalmia would at once render the case highly communicable. The relapse on board ship of an old trachoma, previously quiescent, sometimes infects other passengers. There are recorded instances which prove this. The average case of trachoma found on arrival here, however, will usually show evidences of long standing if a very careful examination is made of the entire conjunctival surface after subsidence of the more acute symptoms. The raspberry-like appearance assumed by the conjunctiva during a relapse is quite analogous to acute granulations, and only after subsidence of the more acute symptoms can an estimate be made of the length of time the disease has existed. It is this proneness to exacerbations and remissions that makes it such a difficult matter to decide when a case of trachoma has really recovered. When the disease has reached that stage of cicatrization in which the conjunctiva presents that smooth, glistening appearance we are agreed it is cured. This cure, however, is often attained after the patient has suffered from the disease for a long time, only to remain a burden to himself and friends as the result of the various sequelae. Experience has shown that, in order to ascertain definitely whether or not trachoma is present, it is necessary to bring clearly into view the retrotarsal fold by doubly everting the upper lid. This is best accomplished by the use of a small instrument.

Since about three-fourths of arriving aliens are medically examined at Ellis Island, a brief description of the method of detecting and certifying trachoma there will be given. The immigrants pass in line, several feet apart, before the medical examiners. One doctor looks especially for mental conditions, another for general diseases and defects, while the last doctor examines with special reference to disease of the eyes. Both eyelids are everted with the assistance of some small blunt-pointed instrument, in such a manner that the retrotarsal folds are clearly seen. Those with any abnormal conditions are put aside for further examination and diagnosis. In the rooms reserved for this purpose the alien is carefully examined. If the eye has trachoma, or is thought to be suspicious, the alien is sent to the Immigrant Hospital for treatment and diagnosis. Apparently trachoma commences as an acute conjunctivitis, and while this inflammatory condition may vary somewhat from the average acute conjunctivitis, it requires observation and treatment varying from several days to several weeks before a positive diagnosis can be made. This rule is followed in the medical examination of aliens, and it is found that the ordinary catarrhal conjunctivitis will subside in a comparatively short time, while the trachomatous case will soon show the hypertrophy and present the raspberry-like appearance so characteristic of acute trachoma.

The diagnosis and prognosis of trachoma is the most troublesome subject with which the medical examiner of aliens has to deal. The diagnosis of a well-marked case presents no special difficulty to one at all familiar with the disease, but the borderland cases, those of long standing and quiescent at the time of examination, or too acutely inflamed to classify, are the difficult cases to decide. While the disease is communicable from the beginning, just at what stage of the process of cicatrization the line is to be drawn between contagious and noncontagious, is one of the difficult problems in connection with the certification of trachoma under the immigration law. If we agree that the disease is not communicable when there is no secretion present, who can say when this quiet stage will give way to a condition analogous to acute granulations under the stimulus of some foreign substance and be communicable? It must be remembered that when the diagnosis of trachoma is made in the case of an alien and he is so certified to the immigration authorities, he is also certified as having a "dangerous contagious disease," which puts him within the provisions of the law, thus making his deportation mandatory. This is a serious matter with the alien, who has probably sold all of his worldly goods to secure enough money to come to America. It is therefore of the highest importance that there be no mistake in the diagnosis. A mistake made in the diagnosis of this disease by the general practitioner works no special hardship on the patient, but sometimes enables him to make a quick cure in his case of trachoma (?).

Trachoma is essentially a chronic disease, the time from the stage of invasion to the terminal one of cicatrization is often one of years, and quick cures are usually mistaken diagnoses. Observation and treatment are necessary in acute cases and relapses. Simple remedies will cure the acute catarrhal conjunctivitis in a reasonable time, but will not affect the trachoma. Follicular conjunctivitis resembles the granular form of the disease. The follicles, however, are arranged in rows not unlike small beads, and their distribution corresponds with the normal follicles of the conjunctiva. They cause comparatively little trouble and recover without damage to the conjunctiva. The pavement-like epithelium of vernal catarrh, covered with a milky film and characterized by excessive itching, burning and watering of the eyes, and coming on with the first warm days of spring, will usually clear up the diagnosis.

Parinaud's conjunctivitis, rather a rare disease, said to simulate a severe trachoma, is ordinarily unilateral, affects the neighboring lymph-nodes and has marked constitutional symptoms.

There is plenty of evidence to prove that trachoma is a communicable disease; the condition of the eyes, varying from defective vision to total blindness, often seen as a result of this disease, shows that it must be considered dangerous, not directly to life, but certainly to the sense of sight, by reason of the destructive effect on the tissues of the eye. Surely any disease which will destroy the sense of sight must be considered a dangerous disease.

The number of persons in foreign countries who are not permitted to embark for this country because they have trachoma must be constantly increasing. Their relatives and friends are arriving here in a constant stream and are, of course, much interested in gaining the admission of those who are now prevented from emigrating here. Every effort is made to enlist the public sympathy with this end in view, and the daily papers very often contain minute descriptions of the many hardships of deportations. The diseased members of a family are often left behind, while those able to pass our examinations come to this country and establish a residence. When the diseased alien arrives it is then pointed

V. 6
1913

out that to deport him would be a great hardship to separate families, as all of the family are in this country. Deportation is unquestionably a hardship, and so is isolation in small-pox, but who would say that the latter should mingle with others because of the dread of the pest-house? One had better have small-pox than severe trachoma. At various times there have been discussions as to whether there should be some modification in the government's classification of this disease. Before any such change is determined on, however, it is well to consider that any such modification would mean the addition to our population of these thousands of trachomatous aliens whose emigration to this country is now prohibited.

It is a fact that trachoma is more easily preventable than curable, and is a splendid example of the old adage, "an ounce of prevention is worth a pound of cure." Our immigration law is the only guard we have against a disease that causes visual damage in 75 per cent. of untreated cases. Aside from the individual hardships and the ever-present handicap of the victims of this disease, there is an economic side that must be considered. The desperate conditions that may result from trachoma will impose financial burdens on the community and blindness may render its victims public charges. Every deportation probably entails some hardship, but deportations will occur as long as we have an immigration law. The sad cases are unfortunately not a few, but this is inevitable, and we should not allow this to work on our sympathy to such an extent that we are blinded to our duty to our own people. The fact that trachoma is already present is no argument in favor of importing more. The plea is often made by relatives and friends that these unfortunate people be sent to local hospitals for treatment at their own expense, and the law has provision for certain cases to be so treated. They are, however, usually not satisfied to remain there until a cure has been effected, and the doctor is continually importuned to release them. In fact, it is difficult to say when a disease is actually eradicated which is so prone to relapse as is trachoma. Although trachoma is very resistant to treatment, it is curable, and those aliens suffering from it should look to their treatment and cure before presenting themselves for admission to this country. There are plenty of immigrants seeking entry to our country who are free from disease, and there is no reason why we should accept those suffering from trachoma, a dangerous communicable disease. While trachoma is excluded as a "dangerous contagious disease" by the general government, there has so far been apparently little notice taken of it by the various states. Although many observers have sought for the cause of trachoma, its etiology is still in doubt, and we are unfortunately compelled to rely on clinical manifestations for diagnosis. Trachoma is a public health problem of the highest importance, and too much consideration cannot be given to its control.

TRACHOMA AMONG THE INDIANS

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The destructive effects of trachoma on the eyesight and the difficulty and tediousness of effecting a cure are so well known that the apparent rarity of the disease in continental America has, until recently, been a cause for congratulation.

It is true that, prior to 1897, cases of trachoma in recently landed immigrants were common enough in the eye clinics of our large cities. Since the time, however, when trachoma was classed by the federal authorities as a dangerous contagious disease, the exclusion of aliens so afflicted has been made mandatory by law and every arriving alien is examined to determine the presence or the absence of the disease in question.

As trachoma does not originate, *de novo*, in any uninfected territory, and its presence in any locality can be ascribed to previous importation, the action of the government in excluding persons afflicted with a communicable disease so likely to result in reduced social and economic efficiency has been a public-health measure of the first order. This measure has given our citizens the assurance that the members of those afflicted with this destructive disease will not, at present, receive many additions from other lands.

Recent investigations have shown, however, that there exist in the United States a number of important endemic foci of trachoma, among them the Indians, and it is the purpose of this paper to deal with the prevalence of this disease in that race. The facts herewith presented are based on the results of an investigation made last autumn by the Public Health Service as to the prevalence of tuberculosis, trachoma, small-pox and other infectious and contagious diseases among the various Indian tribes of the United States. The investigation was ordered by Congress and a report of the findings was rendered to that body Jan. 25, 1913, and published as Senate Document 1038.

The investigation was conducted under the immediate supervision of Assistant Surgeon-General J. W. Kerr, and I was one of the fourteen officers of the Public Health Service who took part in the inquiry.

As a result of the investigation, it may be stated at once that trachoma was found to be a veritable scourge among the Indians, as will be amply shown by the figures herewith presented.

It should be mentioned that the Indian Service had already called the attention of Congress and the medical world to the prevalence of this disease among Indians, and the investigation made by the Public Health Service was the direct outcome of such reports.

SCOPE OF THE INVESTIGATION

The Indian population of the United States is widely dispersed, individuals of this race being located in practically every state, and Indian reservations being situated in at least twenty-five states. According to official figures of the Indian Office, the Indian population in the continental United States is 322,715.

Of this number 39,231, or approximately one-eighth, were examined by the officers engaged in the investigation. Considerable difficulty was encountered in making these examinations because of the season in which the investigation was begun (fall of 1912), the short time available (a full report of the investigation had to be rendered to Congress before Feb. 1, 1913), the wide areas over which Indians are scattered on reservations and the inaccessibility of places where Indians are located. Long distances were traveled by the several investigators on foot, on horseback, by teams, motor-cars and motor-boats.

Besides the physical difficulties just mentioned, the natural diffidence or distrust of the Indians had to be overcome. Many Indians object to furnishing information about themselves and are loath to permit physical examination, either because they are ashamed of bodily defects or diseases or fear to subject themselves to malign influences.

RESULTS OF THE INVESTIGATION

Out of the 39,231 Indians examined, 8,940 individuals, or 22.7 per cent. of the entire number examined, were found to have trachoma. If this rate of infection were found to prevail for the entire Indian population of the United States, there are, at the present time, some 72,000 cases of this disease among this class of the population.

The persons examined represented both sexes and all ages, and included students in Indian boarding-schools, in day-schools and mission schools, and reservation Indians.

The accompanying table shows the number of Indians examined in each state, the number of cases of trachoma found, and the percentage of incidence.

TABLE SHOWING PREVALENCE OF TRACHOMA AMONG INDIANS IN DIFFERENT PARTS OF COUNTRY

State	Indians Examined No.	Cases of Trachoma	Per Cent.
Arizona	5,873	1,459	24.9
California	1,555	238	15.3
Colorado	292	41	15.64
Florida	22	....	....
Idaho	5296	84	15.96
Iowa	53	17	32.04
Kansas	834	176	21 1
Michigan	643	48	7.46
Minnesota	3,542	533	15.05
Montana	2,042	537	26.3
Nebraska	322	130	41
Nevada	851	229	26.9
New Mexico	2,207	494	22.38
New York	943	2	0.2
North Carolina	317	23	7
North Dakota	3,447	791	22.94
Oklahoma	3,252	2,235	68.72
Oregon	904	94	10.4
Pennsylvania	552	76	13.76
South Dakota	6,121	1,059	17.24
Utah	182	75	39
Virginia	43	13	30.2
Washington	1,347	180	13.85
Wisconsin	2,999	207	6.86
Wyoming	392	199	51
	39,231	8,940	22.7

It will be seen from the table that, while the general rate of trachoma infection was found to be 22.7 per cent., the incidence of this disease varies greatly among Indians in different sections of the country; it varies, moreover (this the table does not show), even in different parts of the same reservation.

In Oklahoma the rate of infection found was excessively high. Among 3,252 Indians examined, 2,237, or 68.7 per cent., were found to be trachomatous. In New York, on the other hand, only two, or 0.2 per cent., of the 943 Indians examined had the disease, and it was found that these individuals had contracted the infection during their stay at an Indian boarding-school.

Other states which presented a high rate of prevalence among the Indian population, were: Wyoming, with 51 per cent.; Nebraska, with 41 per cent.; Utah, with 39 per cent.; Arizona, with 24 per cent., and New Mexico with 22.38 per cent.

PREVALENCE OF TRACHOMA IN INDIAN BOARDING-SCHOOLS

The prevalence of trachoma in Indian boarding-schools was naturally deemed of the highest importance in these investigations. The federal government looks on the boarding-school as its chief agency in raising the Indian's plane of civilization and in fitting him for citizenship. The conditions inseparable from institutional life are so favorable to the propagation of communicable disease that the question of the prevalence of trachoma in Indian boarding-schools was carefully gone into.

Data from 133 of these schools were collected and tabulated with the following results:

Out of 16,470 pupils in Indian boarding-schools, 4,916 cases of trachoma were found, a general percentage of 29.86. The highest percentage found was reported at the Rainey Mountain School in Oklahoma, where, out of 114 pupils examined, no less than 105, or 92.10 per cent. were found to be trachomatous, and the incidence of trachoma in the Indian boarding-schools of this state was found to be 69.13 per cent. Only three schools, of the entire number visited, one in Wisconsin and two in New York, were found to be free from trachoma. This is accounted for by the fact that no trachoma was found in the Indian population from which the inmates of these schools are drawn.

Classifying the schools according to the percentage of trachoma, it was found that in two schools over 90 per cent. of the pupils were trachomatous; in eight from 80 to 90 per cent.; in ten, from 70 to 80 per cent.; in nine, from 60 to 70 per cent.; in eight, from 50 to 60 per cent.; in eight, from 40 to 50 per cent.; in seven from 35 to 40 per cent.; in twelve, from 30 to 35 per cent.; in fourteen, from 25 to 30 per cent.; in ten, from 20 to 25 per cent.; in seventeen, from 15 to 20 per cent.; in eleven, from 10 to 15 per cent.; in ten, from 5 to 10 per cent.; and in four, from 0.5 to 5 per cent. In three schools only was no trachoma found. In eighty-eight schools, or 66 per cent., of the total number examined, 20 per cent., or more, of the pupils were found suffering from trachoma.

The percentage of trachoma incidence in boarding-school pupils, in general, 29.86 per cent., is considerably higher than that found for the total number examined (22.7 per cent.). The rate was also observed, in reservation boarding-schools, generally to be higher than in the reservations from which the pupils are drawn; while in the non-reservation boarding-schools, which are not located in reservations, but receive Indians from various parts of the country, it was found that groups of pupils from areas where trachoma is absent, or but slightly prevalent, showed a high rate of infection.

We are justified therefore in inferring that the Indian boarding-school has been an important factor in the dissemination of trachoma among the Indians.

PREVALENCE OF TRACHOMA IN INDIAN DAY-SCHOOLS

In addition to reservation and non-reservation boarding-schools, the government maintains Indian day-schools on the several reservations. The incidence of trachoma in day-school pupils was also investigated. The data collected were not so complete as those for boarding-schools because many of the camps were visited on days when the day-schools were not in session and others were located in inaccessible situations.

Nevertheless 3,488 day-pupils were examined and 752 cases of trachoma were found, a percentage of 21.55. The Indian day-pupils of Arizona were found to show the highest incidence, 41.3 per cent. of 990 Indian day-school children suffering from the disease. This is greatly in excess of the general rate (24.9 per cent.) found for the state.

On the other hand, in New York, out of 335 pupils examined, none had the disease. In no other state where Indians were examined were the day-school children free from trachoma.

If we exclude from the total just mentioned the relatively large proportion of New York day-schools pupils in whom no trachoma was present, the rate of prevalence, exclusive of this state, works out at 24 per cent.

This is in excess of the general average found for the total examination, but is considerably below the percentage found in boarding-schools.

The prevalence of trachoma in Indian day-schools seems to correspond fairly well with the general rate for reservations on which they are situated, and serves further to strengthen the inference that the higher rate of trachoma found in Indian boarding-schools is due to its spread among the inmates of those institutions.

PREVALENCE OF TRACHOMA AMONG RESERVATION INDIANS

Reservation Indians are considered to be Indians living on reservations and not examined in boarding-schools or day-schools. Their number included children below the school age and adults. During the course of the investigation 17,822 Indians of this class were examined with the result that 3,064, or 17.2 per cent., were found to be afflicted with trachoma. The majority of these individuals were examined either in the course of house-to-house inspection, or advantage was taken of gatherings at agencies and subagencies for the distribution of rations and the like.

The prevalence of trachoma in this group is seen to be distinctly below the rate found for the total number examined, the boarding-school rate and the day-school rate. On the other hand, by reason of the greater average age of the individuals and the consequent longer duration of the disease, most of the instances of visual damage were found among their number. The percentage of trachoma incidence noted for the various states varied from 60.40 per cent. in Oklahoma to 0 for New York and Florida. Other states with a high rate of prevalence were Utah, with 39.2 per cent.; Wyoming, with 37.26 per cent.; Nevada, with 36.5 per cent., and Montana with 30.76 per cent. It is worth noting that, in this state, the rate of prevalence among reservation Indians was found to exceed that in the boarding-schools and day-schools. States with a low rate of prevalence were Michigan, with 1.21 per cent.; Wisconsin, with 4.39 per cent., and North Carolina, with 5.45 per cent.

It is evident, as a result of these investigations, that there is a very high rate of trachoma prevalence among nearly all the Indians of the country.

INCIDENCE OF TRACHOMA ACCORDING TO AGE, SEX, AND DEGREE OF INDIAN BLOOD

It may be of interest to give a few figures as to the incidence of the disease according to the age, sex, and degree of Indian blood of those affected.

Age.—Of over 2,000 cases of trachoma in a group of 10,425 Indians examined, in which records with respect to age were kept and compiled, 4.5 per cent. were under 6; over 50 per cent. between 6 and 20 years of age, and the remainder over 20 years of age. It is evident from the preceding that those of school age are the greatest sufferers. The disease is very rare in infants under 1 year of age yet a few cases were found among babies less than 12 months old.

Sex.—In this same group of 10,425 the incidence was found to be slightly higher in females than in males. Of this total number 5,303 were males with 984 cases of trachoma, and 5,122 were females with 1,083 cases. The somewhat greater incidence among the females is only natural in view of the more domestic life of women and their more intimate association with the members of the family.

Degree of Indian Blood.—From the observations made, it was evident that the incidence of trachoma was greater among the full-bloods than among the mixed-bloods examined. This may be attributed, however, to the more prosperous circumstances and the better living conditions which obtain among the mixed-bloods.

VISUAL DAMAGE DUE TO TRACHOMA

A complete census would have been necessary to determine the total amount of blindness due to trachoma among the Indians. Certain observations, however, were made and compiled in the case of 23,560 Indians examined. Of this number 5,505 or 23.4 per cent. had trachoma of which 527 showed, at that time, marked damage to vision. These injuries were of the usual type and consisted variously of corneal ulcers, corneal opacities, pannus, entropion with keratitis, perforation of the cornea with resulting evacuation of the contents of the globe, etc. In 141 individuals the damage consisted of blindness in one or both eyes.

These figures by no means represent the extent of visual damage among the Indians due to trachoma. It should be borne in mind that, in relatively few cases, among those of school age, had the disease progressed sufficiently to produce corneal complications, and the schoolchildren, numerically, were the largest class examined. Moreover, Indians suffering from marked visual disturbance were least accessible to examination and some of these unfortunates evaded the examiners because of their sensitiveness in regard to physical deformities, particularly blindness.

At least 90 per cent. of the complications found in the Indians I examined were observed in the reservation Indians, exclusive of school-children. This is only natural in view of the long duration and chronicity of the disease.

With respect to the types of the disorder encountered, a wide variation was observed, many of the cases being mild and easily amenable to treatment, while others were of the utmost possible severity.

ORIGIN AND DURATION OF TRACHOMA AMONG THE INDIANS

The length of time trachoma has existed among the Indians is purely a matter of speculation.

The finding of cases in which cicatricial tissue only was present, the fact that aged Indians with trachoma stated that they had suffered from "sore eyes" all their lives, the history given by some of having contracted the disease from a subject long since dead, lead us to suppose that the disease has been among them for at least a generation.

Clark is of the opinion that trachoma is doubtless of more recent origin than tuberculosis among the Indians, but has existed for many years.

So, while there is no question but that the disease is widely disseminated among the Indians and has existed for years, the source and date of its introduction must be conjectural.

INSANITARY CONDITIONS AMONG THE INDIANS AND THEIR BEARING ON THE PREVALENCE OF TRACHOMA

Climatic Influences.—Generally speaking, Indians inhabit healthful sections of the country, and the large majority of Indian reservations are situated where the soil is fertile, the elevation high and the percentage of sunny days great throughout the year.

The scanty annual rainfall, however, and the high winds lead to the occurrence of numerous dust storms. Added to this is the activity of the sunlight due to the great percentage of sunny days, the lack of trees, and the greater content of ultraviolet rays by reason of the

high elevation of most Indian reservations. Many Indians were encountered during the investigations whose eyes were sensitive to light and who sought protection by the use of smoked glasses. The irritation due to excessive light and dust leads to the frequent rubbing of the eyes with the fingers, thus rendering easy the transfer of infection from fingers to eyes.

The excessive severity of the winters also contributes to the dissemination of trachoma, as the Indian remains indoors a great deal during the cold season, in order to keep warm, and the personal contacts in his crowded cabin are prolonged and intimate.

Housing Conditions.—In his savage state the Indian lived an open-air, nomadic life and his tepees, while often dirty, were well ventilated.

Since the middle of the nineteenth century, however, the Indians have been confined to reservations and constrained to live in houses. They are still generally ignorant of the elementary principles of domestic hygiene required by a stationary abode. This does not apply, however, to all Indians, many of whom were found to be of unusual intelligence and good sanitary habits.

Their houses together with their domestic and social habits, however, play an important part in the dissemination of trachoma. The typical Indian house is a small, one-roomed frame or log structure, which, in over one-half the instances has a dirt floor. The average Indian family is large, four or five children being by no means uncommon. Personal privacy is generally unknown, and all live and sleep together in the crowded cabin whose doors and windows are carefully shut in the winter, for warmth, the windows being frequently nailed shut. Whatever washing of the hands and face takes place is done in a common wash-basin, and towels, when present, are used by all members of the family. The bed-clothing, consisting of quilts and blankets, is used, without washing, until worn out, and indiscriminately by the various members of the household. The scanty accommodations of the crowded cabin are likely to be further strained by the advent of visitors who, with true Indian hospitality, are welcomed and may stay days or weeks.

Discharges from the eyes and nose are usually removed by the fingers, which are inadequately wiped on the clothing or any convenient object. It was no unusual sight to see a trachomatous mother removing the secretion from the corners of her eyes with her fingers, and then endeavoring to assist the examiner in examining the eyes of her children.

Add, in the summer time the presence of numerous flies, and the sociable nature of the Indian which delights in visits and social gatherings of all kinds, and the wide-spread prevalence of trachoma among the Indians is readily accounted for.

THE PUBLIC HEALTH SIGNIFICANCE OF THE PRESENCE OF TRACHOMA AMONG THE INDIANS

The public-health significance of the presence of trachoma among the Indians has now become of considerable importance.

In former years the Indians were closely confined to their reservations and their intercourse with the white race was scant. The presence of trachoma among the Indians, at that time, while deplorable, wrought no injury to others than themselves. At present, conditions are about to be greatly changed. County after county on Indian reservations is being opened to white settlement, and the day seems not far distant when the Indian will be merged in the general population. An infection, therefore, which menaces one of the essential senses of the body seems on the eve of being widely spread through territory in which, at present, it is confined to restricted areas.

In justice to the officers of the Indian service it should be stated that they are cognizant of these conditions and are doing what they can to control them. They have been hampered by lack of men and money, but have asked for liberal appropriations to control the spread of trachoma and treat those so afflicted. It is to be hoped that such funds will be forthcoming in the near future. In the meantime they have placed what men they could in the field to treat eye diseases among the Indians, and agency and school physicians in general are on the watch for the disease and treat as many of those afflicted as they can.

CONCLUSIONS

1. Trachoma is exceedingly prevalent among the Indians, the prevalence being (a) highest in Indian boarding-schools, (b) less in Indian day-schools, and (c) least among reservation Indians above and below school-age.

2. The origin and duration of the infection among the Indians is unknown, but its wide dissemination is readily accounted for by the housing conditions and want of the knowledge of personal hygiene among them.

3. The opening of reservations to white settlement and the foreshadowed absorption of the Indian by the white population renders the present wide-spread diffusion of trachoma among the Indians a serious menace to future white populations of Indian reservations.

TRACHOMA AMONG THE NATIVES OF THE MOUNTAINS OF EASTERN KENTUCKY

J. A. STUCKY, M.D.
LEXINGTON, KY.

For more than twenty-five years, a large and rapidly increasing number of cases of trachoma, with many of the devastating sight-destroying sequelae, have been coming to me from the mountains of eastern Kentucky. I was impressed with the genuineness, the simplicity, honesty and sincerity of the large majority of those so afflicted—and the fact that very few of them had the first rudiments of a common school education, indeed, few of them could read or write, and many were unfamiliar with the alphabet. Often I would see the patients only once—there being no funds to maintain them or provision of any kind available for keeping them in the hospital for treatment. After a few months many of them would return with complete destruction of the eye or with impaired vision beyond restoration.

Besides this class of cases many were brought with most extensive and complicated suppuration and destruction of the middle ear and mastoid process, with systemic, meningeal, auditory and psychic sequelae—also cases of pansinusitis of the nasal accessory sinuses, some with involvement of the anterior frontal lobe (the silent area) of the brain.

All of these cases came from an area in the heart of the mountains composed of seven counties. After investigation I found most of these counties were so-called "pauper counties"—with no money in the county treasury, and not so much as an almshouse. My "scien-

tific curiosity" was aroused as to the source of the infection, the conditions and surroundings under which these people lived, and in the physical and sanitary conditions of the people with whom they associated. My first trip of investigation, lasting four days, was made in the fall of 1910. Going over the Lexington and Eastern railway to a station 84 miles away from my home, I went muleback 27 miles into the interior of the mountains and held a clinic for two days. The following April (1911) I established a clinic at Hindman, Knott Co., Ky., which was then 45 miles from the railroad, making the trip on muleback, accompanied by two nurses, a guide and a pack mule to carry all our clothing, hospital supplies and instruments for a four days' clinic. It took nearly two days to make the trip, much of which could not be adequately described.

Three elements which constantly presented themselves on this trip, and which kept us on the qui vive from the start to the finish were the element of (1) uncertainty, (2) expectancy, (3) danger. More detail cannot be given than this at this time, but this much may be said that my "scientific curiosity" and desire for investigation was liberally paid for in these three elements, in the hard, rough, grand and many times enchanting journey.

The clinic was founded in connection with the W. C. T. U. Settlement School established at Hindman, one

Figs. 1 and 2.—Groups of trachoma patients.

of the most remote and inaccessible parts of the mountains, and has been conducted semiannually since. As the result of examinations and conditions found among the natives, who in large numbers attended the clinics, I made my first report to the American Academy of Ophthalmology and Oto-Laryngology at Indianapolis, September, 1911, at the same time exhibiting a number of photographs taken at the clinic.

As the result of the discussions and suggestions made at the time, I later made the same report and exhibit of photographs to our State Board of Health. At the annual meeting, in Louisville, of the sanitary and health officers of the cities, towns and counties of Kentucky, I was appointed to represent the state, in connection with Dr. John McMullen of the U. S. Bureau of Public Health, to investigate the infectious diseases of the eye and other sanitary conditions in the mountains. Our reports have been made and published in Bulletin 101 of the Bureau of Public Health reports. I presume that it is because of this report that the Section devotes this time to a symposium and discussion of the most prevalent, infectious and dangerous disease of trachoma, found in such large numbers among the natives of the mountains of eastern Kentucky.

In order better to appreciate the report of the U. S. Bureau of Public Health and to accentuate the importance of the situation and condition a very brief description of the people and their method of living must be given. Emerson Hough[1] correctly gives the desired information — though the location of the Hindman clinic, where my observations were made, is fully forty miles further into the interior or heart of the hills. Hough says:

In the Cumberland Mountains of Kentucky there are no roads; the only viaducts are aqueducts, the creekbeds, which have known no shaping by the hands of man. When the bottom of the creekbed becomes impassable, the trail climbs over the shoulder of the bank. Sometimes it crosses from one creek valley over a summit into another valley, and so works its way back sinuously into the hills; a narrow trough, it has no culverts, no bridges, no drainage. When you bog down, you get a rail and hoist out your wagon.

Here live people of arrested development who have always remained Americans. Since the days of Daniel Boone they have been thus unchanged—not laborers, operatives, or salaried folk, but soil-owners and home-makers. These are by no means the "poor whites" of the South, by no means the shiftless Cracker sort, but a keen, bold breed of men, who remain

of the Revolutionary type even to-day. These Americans belong to us, and we owe much to them. There are half a million of them waiting to be discovered as American citizens.

You can go back into the Cumberland country and find the trail where Boone crossed, following the waters, and see that region pretty much as he found it and as he left it, a country with a history arrested, unwritten, waiting. The log cabin is the universal form of the home. You can find to-day the loom, the spinning-wheel, the lard-kettle, the candle-mold, the squirrel-rifle, unchanged in a hundred years. The women there still wear sunbonnets, and they still ride behind, horseback, with their lords and masters; still stand waiting at table while their lords and masters eat.

Strange old Elizabethan phrases of speech still linger. Hot bread still prevails, three meals a day, and pork when they can get it. The men do not go west to pick up prairie lands, but toil laboriously with the ax on steep mountainsides to open the land, plow it precariously with single-shovel plows, or till it laboriously with hoes—men, women and children. Farm machinery is unknown.

You cannot believe, and surely cannot understand, these things without seeing them—cannot believe that this backward country is in America as you know it. But, having seen them,

1. Hough: American Magazine, December, 1912.

you understand why these side-tracked highlanders, lost for more than a century, do not send their children to school. There are no schools, because there are no roads; no roads, because there are no taxes; no taxes, because there is no money; and, coming round again in a circle, no possible interchange of commodities, because there are no roads. There lies the wilderness of the old crossing-place of the Alleghanies as Boone saw it and left it.

In this country you still see the old American type. The men are tall and sinewy, for the most part quiet in habit, slow of speech. Though marrying young and soon aged by hard work, the women are delicately beautiful of face and figure, the children being especially very beautiful. They have nothing of the vacuous "poor white" look, and have not the slightest resemblance to the stolid peasants of Europe.

You will find grown men, of powerful body and powerful mind, with an exactness of estimate, a breadth and justice of mental view, simply astonishing; yet perhaps their total book knowledge will not equal that of a 5-year-old child in communities more fortunate. It is a state of affairs singular and almost unbelievable in our country. I call it a white bondage for lack of a better term. It is simply arrested civilization. Have a look at the population we meantime have imported from the rest of the world in place of these!

In these little cabin homes and rude shacks, windowless, with no opening to the single room but the one door,

Fig. 3.—A group of trachoma cases with corneal and lid complications.

with a lean-to for a chimney or piece of stove-pipe thrust through the side or roof, live the entire family of from eight to twenty. In this room they eat, sleep, cook — live. They all use the same large family towel for days, and this undoubtedly is one of the greatest sources of infection. The method of living is suitable for the propagation of any contagious or infectious disease.

In none of my clinics and on none of my tours of investigation have I met very many cases that I could with confidence diagnose as acute trachomatous infection. The disease, as I have found it, has been chronic, sometimes with months or years intervening between the time of its incipiency and the terminal stage of cicatrization with one or several of the inevitable sequelae. Omitting reference to the initial stage, I have found and treated hundreds of cases of the second and third stage.

In the second stage the palpebral conjunctiva is not only hypertrophied but often studded with granular masses, or granules, undergoing destruction which causes a rough and uneven surface, so characteristic of the disease. It is in this stage that the tarsal cartilage becomes thickened, that newly formed blood-vessels appear in the cornea, resulting in vascular keratitis or pannus, ulceration and trichiasis, and then begins the real suffering complicated with impaired vision, photophobia, lacrimation and discharge of mucoid material which later becomes mucopurulent.

The third stage I found in large numbers of the natives where connective tissue has replaced the palpebral conjunctiva, the tarsal cartilage becomes atrophied (in many instances almost completely destroyed), the result being that the lid is no longer held in its normal position, and the lashes are turned inward and upward, further irritating the already inflamed cornea, thus giving the appearance of frosted ground glass. It is in this stage that the ulcerations of the cornea assume a virulent condition, frequently perforating and emptying the anterior chamber, terminating in adhesions of the iris and lens to the cicatrized cornea. This is one of the sequelae of the disease that I have found for which our science and art offers no hope of amelioration or relief. A much larger number of cases than those just referred to are found, in which from one to two-thirds of the

Fig. 4.—"Too late"—the husband blind from trachoma and the wife with only one-third vision, from the same cause.

posterior palpebral surface is firmly adherent to the eyeball, a condition known as symblepharon. While this is more frequently met in those past middle age who have had trachoma since childhood I have records of twenty-seven cases in which the patients were under 30 years of age.

There can be no doubt of the infectiousness of the disease. Extremely rarely did I find only one case in a family. The nature and method of preparing the food, everything being cooked in grease, or boiled — the large quantity of sorghum molasses and hot soggy bread consumed — all tend to impair digestion and cause vicious changes in metabolism, which lower the vitality and body resistance, and make the inhabitants easy victims to hookworm infection — which the majority of them have. Also the insanitary and unhygienic cabins and huts in which the natives live, large families sleeping huddled together in one windowless room, all of them using for many days the same family towel, which hangs

on the door frame, easily accounts for the spread of the disease.

While I am convinced that it is a communicable disease there exists in some a strange and marked immunity which I cannot explain. Again, in many instances I have met with most active and violent inflammatory exacerbation of the disease in a chronic case, in which one eye only was involved, no disease or "soreness" ever being noticed in the opposite eye. I have met families from three to nine members of which had the disease, while one or two members had slept, eaten and lived for months and years with them, even using the same dirty, trachoma-laden towels to dry their faces and hands, and I have seen other instances in which one eye was dangerously involved with the disease and the opposite eye normal; I am therefore forced to conclude there must be some condition of the conjunctiva in one eye, or in both eyes of some, that creates an immunity to the disease.

I believe that trachoma is due to some organism or bacillus. That it has not yet been discovered and a specific remedy found for its destruction is a reproach to the science and art of bacteriology and ophthalmology.

The disease, as I find it in the mountains of eastern Kentucky, is in no way related to the gonococcus, spirocheta or tubercle bacillus, nor is it carried by flies, because there are surprisingly few flies to be seen.

In the last four years, semiannually riding hundreds of miles on muleback or in a springless mountain wagon, stopping at their humble cabin homes for water or direction as to the right way of going, or sleeping in them when overtaken by night, or when the journey was stopped by the sudden "rising of the creek or river," as the result of a heavy rain which made it unsafe to attempt to ford, I have seen numbers of cases of trachoma, as evidenced by the gummy appearance of the edge of the eyelids, or by ptosis of the upper eyelid, photophobia or profuse lacrimation. The answer to my first question would be "'Twarn't nothin' much, only jes' sore eyes, which git powerful weak at times." The reply to my question, "How long have you had sore eyes?" would be, "Oh, I've follered having sore eyes fur quite a spell, they gits purty nigh well some times, then they busts out wus' than ever."

Such patients do not come to my clinic or seek medical aid, because the pain is not great enough or vision sufficiently impaired to prevent their making the "craps" or "lumberin'" — and live they must. With no knowledge of agriculture or latter-day farming it is hard for the best of them to eke out an existence; they succeed at best in making scant crops because of washed, gullied and unkept soil. Hence they are insufficiently and improperly fed and clothed because they do not know how to prepare and cook food, and have not the means, facilities or opportunities for getting the right kind of clothing for their bodies.

The pathos and tragedy of the condition of the natives in the mountains of eastern Kentucky is that, if they ever receive help of the educated citizen, such help must be carried to them, because they are mountain-locked; are misunderstood and misrepresented, have been crowded back by civilization, and have never "had a fair chance or a square deal."

The conditions as revealed by the facts and figures of the United States government reports of Nov. 8, 1912 (Public Health Bulletin No. 101), are shocking and appalling. This report says that of over 4,000 people examined in five counties 12.5 per cent. had trachoma.

To my semiannual clinics only those patients come with the most painful forms of the disease—those so diseased as to be practically incapacitated from household or other labor. Of the 374 cases seen at the clinic in September, 1912, 113 had trachoma; 40 per cent. had corneal complications; 25 per cent. had photophobia; 10 per cent. had entropion; 2.5 per cent. had symblepharon; 25 per cent. had impaired vision, ranging anywhere from slightly defective vision to total blindness, all these were sequelae of the disease.

The trachomatous patients ranged all the way from small children in mothers' arms to old age. Trachoma patients coming into our ports from Europe are quarantined and forbidden citizenship; and yet, of the 114 of these quarantined cases examined by me, on Ellis Island and in Baltimore, not 0.5 per cent. were comparable in severity to the average of those I see at my clinic in the mountains. Besides the ravages of trachoma, we meet many cases of ear, nose and throat diseases, and probably more of the natives are afflicted with hookworm disease and its sequelae than any other two diseases, and until they are relieved of this there is slow and unsatisfactory response to any medical or surgical treatment for any of their diseases.

Dr. A. Von Sholly, representing the N. Y. Board of Health, who was sent by this board to accompany me on one of my visitations, for the purpose of studying conditions clinically and also to make bacteriologic and microscopic examinations in the acute and chronic stage of the disease, says:

> The situation in this country, at present, is ludicrous—we take precautions to keep out infected aliens, but cherish—at least do very little to discourage—the spread of trachoma in our midst, and worst of all amongst our oldest "American Stock" at that. That trachoma is prevalent in the mountains of eastern Kentucky and in its most destructive form is a deplorable fact, for anyone who goes there, to see. So long as the highways are sewers, the homes and public meeting-places huge cuspidors, and the common wash-basin scantily filled with water and the nearest rag the bathing equipment for the whole family and its guests—not to speak of the common bed, in many instances without sheets or pillow-slips—one cannot be surprised that typhoid fever, tuberculosis, hookworm and trachoma are rampant.

The solution of the problem of eradicating trachoma in the mountains is neither simple nor easy — it is complicated and hard, but I have no doubt that it can be done. The problem can be solved through a threefold agency, under the direction of the State Board of Health and U. S. Bureau of Public Health — this agency to be (1) the medical man, (2) the graduate trained nurse and (3) the schoolteacher. A. Von Sholly says:

> The solution of the problem is dissipation of ignorance by teaching the laws and rules of hygiene. When the belief of the mountain folk that "sore eyes" and "weak eyes" are a visitation of the Lord, to be endured with resignation, is supplanted by the knowledge of how infection is carried and what personal and family hygiene means, there ought to be no difficulty in rooting out the contagion. Illustrated public talks; illustrated pamphlets of instruction; visits to the homes by a trained nurse and social worker who has gained their confidence; clinics, such as those held at Hindman and Buckhorn, in Knott and Perry counties, which cure the early cases and give relief to those with irreparably damaged cornea, foster confidence in the mountain people toward their advisers.

In appealing for aid and cooperation in this work, I have received the reply from medical men that "it is useless for oculists to attempt the medical and surgical treatment of the disease with any satisfactory results

until conditions are changed." In reply to this I want to know who is to change conditions unless it be the medical man, sanitarian and schoolteacher?

With the character of physical and mental "Anglo-Saxon American stock" to deal with in the mountains of Kentucky where trachoma is so prevalent, I feel confident that the disease can be effectually arrested and then eradicated by the methods and system used in the floating ophthalmic hospitals of Egypt,[2] as described and illustrated by photographs.

While we have made no appreciable progress in ascertaining the specific factor in the etiology of the disease, we have made great progress in the treatment. I consider the treatment in certain conditions to be as important as that of virulent ophthalmia, and require almost as active day and night care and judgment in the use of remedies, irrigations and fomentations. I have no hesitancy in saying that for the past three or four years in cases of trachoma, especially those with corneal complications, I accomplished more in two or three weeks with the case in the hospital, than I formerly did in as many months having the patient treat himself at home, and visit me at my office or clinic several times a week.

The old routine method of an occasional application to the everted eyelids and certain remedies to be instilled into the eye several times daily by the patient or relatives is unscientific and unsatisfactory. I know of no infection more tenacious, stubborn, uncertain and harder to eradicate than that of trachoma — nor one that requires closer observation and more careful handling, in order to save vision when impaired by ulceration of the cornea or pannus. For this reason it is essentially a case for hospital treatment and care from a humane, economic and scientific standpoint.

Probably no state has a more efficient board of health than has Kentucky, certainly none with a more competent executive secretary. Who has accomplished more for the members of this great American Medical Association than Dr. J. N. McCormack of Bowling Green, Ky.? But with the very meager financial aid at its disposal, and with no hope of an increase in the appropriation for medical and sanitary purposes in the near future, because of the state's great indebtedness, our State Board of Health has been able to give but little assistance toward the relief of the situation; hence the appeal to the U. S. Bureau of Public Health, which was promptly and most efficiently given, and whose report, already referred to, has caused such widespread interest and comment in medical journals, magazines and newspapers. Within the last three months daily trains are running through the region of this hotbed of trachoma, and the danger of the infection being carried to other towns and cities is being greatly increased.

The condition confronting the federal and state governments is one of more than intensely scientific interest — it is at once humane and economic and should not be viewed from either a political, sectarian or sectional point of view.

The result of the semi-annual clinics held in connection with the W. C. T. U. Settlement School has been most gratifying. The removal of adenoids and tonsils, the treatment given for hookworm, and active prophylactic remedial and surgical means used for the arrest and cure of trachoma, the correction of refractive errors in a very large number, especially those having trachoma, the great improvement in the general physical development and health as well as the changed mental condition of the children and their homes, is remarkable and a most hopeful indication of what could be accomplished were the work done in a more systematic and persistent manner, with improved facilities and equipment, which are sadly lacking in the cabin hospital at Hindman.

Too much credit cannot be given to the faculty and the resident district nurse, and the Board of Health at Hindman. The good work accomplished by Miss Butler, the resident nurse, in caring for infected patients and carrying out after-treatment, as well as her painstaking and tactful method of disseminating knowledge of sanitation, hygiene and the cause and methods through which infectious diseases are transmitted, is a forceful illustration of the claims which I have made for the graduate nurse.[3]

The pictures from which the illustrations were made were taken with a kodak by one of my nurses inexperienced in the use of a camera, but they give a more correct idea of the people, the disease and the conditions than I could convey by pen or speech.

2. Campbell, A. F.: Ophth. Rec., December, 1910.

3. Stucky, J. A.: Another Field of Usefulness for the Graduate Nurse, Pub. Health Nurse Quarterly, January, 1913.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. M'MULLEN, SCHERESCHEWSKY AND STUCKY

DR. EDGAR S. THOMSON, New York: The two main questions brought up by this group of papers are: first, the necessity of excluding immigrants with trachoma, and second, the management of the disease among the mountaineers of Kentucky and the Indians. It seems hardly necessary at this time to lay much stress on the advisability of excluding immigrants, for its necessity has been so well shown, but I would like to express my approval of the system of fines to the steamship companies and also of the care and thoroughness with which immigrants are examined at Ellis Island, in New York City. In this connection I will read an extract from a letter from the Commissioner of Immigration:

"Between July 1, 1911, and June 30, 1912, there were certified, approximately, 720 cases for trachoma. All except 53 were deported, and an attempt to cure these patients was ordered. Of these, 16 were cured, but the remaining cases have not yet yielded to treatment. Treatment is permitted only in cases in which deportation would result in extreme hardship, as, for instance, in cases in which the immigrant has no one to go to on the other side. The present tendency of the department is to restrict to the lowest possible number those who shall be held for treatment, and as to all others to let the law take its course. Trachoma is classed by the Public Health Service as a dangerous, contagious disease, and all who arrive with it may be deported forthwith. In all cases in which a competent medical examination abroad would have disclosed the existence of trachoma, the steamship company bringing the diseased immigrant over is subject to a fine of $100. In 1902 there was no statute permitting the imposition of such a fine, and the proportion of immigrants who came to this country with trachoma was far greater in those days than it is now. The conditions in the steerage quarters on some of the ships were truly horrible, because the cases brought were bad ones and the diseased immigrants mingled freely with those who were not diseased and used the same towels. Of course, all such cases were deported, but the deportation expenses, being low, did not deter steamship companies from bringing over people with trachoma. Their surgeons used to say that they considered many of these cases to be conjunctivitis, rather than trachoma, but such a confusion of the two diseases has occurred far less frequently since Congress permitted the imposition of the $100 fine. Now we rarely see the very worst cases of trachoma, although there are still brought here many cases in which the disease cannot be eradicated, except through

treatment extending over at least one year. Congress has been urged to increase the fine limit to $200, so that the steamship companies shall be deterred from bringing here any immigrants with trachoma." One of the most vexing questions in connection with the exclusion of immigrants and the necessary hardship to individuals is the question of what constitutes a dangerous case. With the pathology of trachoma in its present unsettled state, we should err on the side of safety by excluding any cases of well-marked follicles, or at least place the case under compulsory treatment until it is cured. The clinical diagnosis of trachoma follicles is a subject on which it is impossible in all cases to be dogmatic, but my own feeling is that the typical large ovoid follicle is of trachomatous origin in the vast majority of cases, and that the wide differences that we see in the course of different cases, some running a comparatively mild course and others developing a severe pannus, depends mainly on the amount of local tissue reaction present, which in turn may depend on constitutional tendencies, superimposed infections, etc. I strongly endorse a point in Dr. McMullen's paper, in which he says, in effect, that a quiescent case may at some subsequent period develop conjunctival secretion and become actively contagious and a serious menace to the community. I am willing to admit the condition of "follicular conjunctivitis," in which fine follicles are scattered all over the tarsus and retrotarsal folds, for these cases have well-marked peculiarities, but as to the mild cases with the pale oval follicle, I am inclined to the supposition that these are mild infections of true trachoma. The follicles of vernal catarrh and the other special forms of follicle have marked clinical characteristics. If such mild cases are to be admitted to the country their treatment must be compulsory. Except in rare instances, such patients leave us before the cure is completed, if the case is at all protracted. The treatment of the mountaineers and Indians must be accomplished in a different way but is a no less serious question. I think everyone agrees that while trachoma is a disease we expect more commonly to find in crowded cities, it is just as virulent in healthful country districts, where unsanitary conditions prevail. It seems to me that the great need, as Dr. Stucky pointed out, is for some sort of hospitals, or at least clinics, in these out of the way districts, where systematic and persistent treatment may finally eradicate the disease. And this applies equally to the problem in the case of the Indian. In some of its phases, trachoma is an exceedingly stubborn disease, prone to relapses and exacerbations and constant supervision of almost every case is necessary. I am strongly in favor of early expression, for I believe that this has a direct bearing on the prevalence of pannus. This is denied in some quarters, where the presence of pannus is looked on as placing the case in a somewhat different category from the ordinary class of cases characterized by follicles. We certainly see vastly fewer cases of pannus in our New York clinics since about ten years ago, when an active campaign was started in the schools and expression of the early cases was done on a large scale. It seems to me that it is not worth while to limit our activities by the discussion of any especially fine pathologic points. The important clinical facts we know and our course and duty in the matter are both clear.

DR. L. WEBSTER FOX, Philadelphia: The paper read by Dr. McMullen might be construed as a plea for a continuation and rigid enforcement of the deportation clause of the immigration law, which provides for the exclusion from admission to the United States of immigrants suffering from trachoma on the ground that it is a dangerous, contagious disease. Dr. McMullen has shown that the proportion of trachoma cases to the total of arriving aliens has considerably diminished in one year. The only cheering ray of hope in this paper for the poor, trachomatous immigrant, is contained in the remark that although trachoma is very resistant to treatment, it is curable. Without commenting on the merits or demerits of the restrictions and penalties of this part of the immigration law, I will relate my own experience with immigrants found, on their arrival at the port of Philadelphia, to be suffering from trachoma, with a view to showing that such cases are not only curable but that a radical cure can be effected within a reasonable period. During the past year I operated on eighty-seven patients suffering from typical trachoma. Among this number forty-four immigrants who were found to be suffering from trachoma, on arriving in the United States, have been transferred across the Delaware River from the Detention Station at Gloucester to the Medico-Chirurgical Hospital in Philadelphia for prompt treatment of their condition by Dr. Oakley of the Marine Hospital Service. These cases would permit of no delay and I have therefore operated on them by the method known as grattage, which almost invariably brings about a radical cure within a reasonable time. I have practiced grattage with great success and satisfaction from the time I saw it performed by Dr. Darier, in Paris, in 1892. The operation requires, first of all, complete eversion of the lids and exposure of the trachomatous conjunctiva into every part of the culdesac. This is easily effected by means of the Darier forceps, which resemble a catch dressing-forceps with three pins on the male blade. The roughened, thickened, congested conjunctiva is next thoroughly scrubbed with a tooth brush dipped in a solution of bichlorid of mercury, 1 to 2,000. With a three-bladed scarificator, the whole of the palpebral conjunctiva is then scarified, and by means of gauze sponges it is rubbed until the whole of the palpebral surface of the lid is perfectly smooth. Although this seems to be a somewhat formidable procedure, it is surprising how quickly the swelling subsides under the use of antiphlogistic and cold applications and the patient is cured of trachoma. All of the forty-four immigrants referred to were discharged as is shown by Dr. Oakley's official report. This method of treating such cases is so radical that recurrence of the trachoma is unusual. In addition to the grattage, it is advisable in some cases to perform Burow's operation, or to excise the tarsus. The results obtained by this line of treatment have been so satisfactory that the individuals referred to, who had traveled many thousands of miles to join their friends and families in this country, are no longer a menace to the health of the community, and have been humanely saved from deportation.

No record of time in two cases.

1 case	in hospital 9 days	5 cases	in hospital 18 days
2 cases	in hospital 10 days	3 cases	in hospital 19 days
1 case	in hospital 12 days	1 case	in hospital 20 days
3 cases	in hospital 13 days	1 case	in hospital 21 days
5 cases	in hospital 14 days	1 case	in hospital 23 days
2 cases	in hospital 15 days	2 cases	in hospital 24 days
4 cases	in hospital 16 days	1 case	in hospital 25 days
		14	
6 cases	in hospital 17 days	26	
26		40	cases released to date, 6/3/13

DR. F. PARK LEWIS, Buffalo, N. Y.: The question of the control of trachoma naturally resolves itself into two parts, the protection of our ports from immigrants, and the protection of those who are already within the country from the ones infected. Four years ago in Naples I made some investigation of the methods then used to prevent immigrants who had trachoma from attempting to leave Italy. I found that the steamship companies refused transportation to five thousand people each year because they had trachoma, knowing that the penalty would be the re-transportation of those infected. I believe we need give little attention to our ports of entry. The United States government is fully alive to the importance of this phase of the subject, and is, I am sure, giving us adequate protection at these points. The vital question is the localization and control of trachoma now in the United States, and the further determination as to whether these centers are broadening, and there seems to be very little doubt in regard to this. In every school for the blind in the United States, except that of Arkansas, the chief cause of blindness is ophthalmia neonatorum. In Arkansas the chief cause is trachoma. The eastern and southern portions of the state are almost free. In the Arkansas School for the Blind there were 199 white and 28 colored pupils enrolled. The last report shows that the number of cases of blindness from trachoma is 115, which demonstrates that in Arkansas and in adjacent Missouri there is an enormous amount of active,

acute trachoma, a thing of great importance to be determined. In the second place, we are finding cases manifesting themselves in various isolated localities. A few years ago it was unusual to find trachoma in western New York. Dr. Kerr said that trachoma among the Indians in New York was in the proportion of 0.2 per cent, and yet I think it is the experience in western New York, among the ophthalmologists, that gradually new, acute cases are appearing from time to time. In Oklahoma the infection among the Indians is rapidly spreading to the white population. The white schools have already become infected, to the extent of more than 25 per cent., as I think Dr. Kerr has shown. Every active case of trachoma should be reported officially to the health departments of the several states. Until the extent of the disease is known we are as helpless as we would be with any other infectious disease, such as small-pox, if it were scattered over areas over which we had no control. In the second place, our treatment of these conditions consists of after-hospital or dispensary care for a large proportion of the cases. The patients with acute cases appear in the hospital and dispensary, then go back to their homes when they again act as sources of infection. Until we can use the social service of various philanthropies in these localities, by following the people into their homes and using measures to prevent infections, we shall not secure control of this menacing disease.

DR. S. D. RISLEY, Philadelphia: Viewed from the sociologic aspect, I recall the fact that only a week or ten days ago I noticed in a newspaper that the strikers in the Kanawha region of West Virginia were being replaced by mountaineers from Kentucky, and if these mountaineers are from the district infected with trachoma, it is important from the hygienic standpoint to control the matter since it might spread from them into the mountains of West Virginia and thence into the population throughout the entire region.

DR. GILBERT E. SEAMAN, Milwaukee, Wis.: The kind of work Dr. Stucky is doing and this kind of a situation are moving illustrations of the fact that the handling of a dangerous, contagious disease is a matter for state or national government. It would of course be foolish to expect a few individuals, led by even as enthusiastic a man as Dr. Stucky and the organization in Kentucky, to cope with this situation without the aid of the government. I did not understand from Dr. Stucky's paper that the state of Kentucky was doing its share in this work. I believe, from a somewhat full experience in a district populated by foreigners, that the inspection at the port of New York and at the other ports is not so effective in keeping out trachoma as we might think. We have, in the city of Milwaukee, a large immigration from southern Europe at the present time. We have not a very great proportion of cases of trachoma, but the cases we do see are among that class of people, and generally among people who have recently come from Europe. I have observed the inspection, not in recent years, but formerly, at the port of New York and at other ports, and I do not think sufficient time is given to the attempt to make a diagnosis. It is not an easy matter to make a diagnosis in the early stages of trachoma, and I believe that many of the cases get through at the ports that might be diagnosed and properly handled and treated if more time were given to the particular matter, and probably more expert observation in some instances. But what I particularly wish to emphasize is that without the help of the government—and when I say the help of the government I mean financial aid and the organization that comes from financial aid—such a situation as that in the mountains of Kentucky cannot be adequately handled. The highest function of government, I believe, is the preservation of the lives and health of its people, and I would not hesitate, if I were Dr. Stucky and so situated, to ask the state of Kentucky to provide all the money necessary and insist on it. I believe the lecture of Dr. Stucky, if delivered to the people of Kentucky and to the legislature in session, followed by a request for funds, would bring forth financial aid. It is, however, a very difficult matter to get sufficient financial aid for proper health work.

DR. WILLIAM ZENTMAYER, Philadelphia: I wish to bear testimony to the effectiveness of the examinations at the port of Philadelphia. Some five years ago my assistant, Dr. Dewey, at the Wills Hospital, took occasion to go over the records for the previous twenty years during which there were from ten to eighteen thousand new eye cases. He found that twenty years ago 9 per cent. of the cases were trachoma, while twenty years later the percentage had fallen to 0.9 of 1 per cent., and that was five years ago. Dr. Risley and myself were on the Committee of "Annual Report" of Wills Hospital this year and we were struck by the infinitesimal number of plastic operations called for by trachoma. It was almost negligible. Then I was very glad to hear Dr. Fox speak of the cure of trachoma. I was taken to task recently in a published article for my optimistic views on trachoma. I had expressed the opinion that in my experience the damage to the eyes from trachoma had rarely been so serious as to incapacitate the patients for work. It may be because of the thorough examination at our ports that we have been fortunate in seeing very mild cases in Philadelphia.

DR. LUCIEN HOWE, Buffalo, N. Y.: I do not think the statement made by Dr. Fox should be allowed to pass unchallenged. We have all practiced grattage, we have all curetted, but I found it practically impossible to obtain a cure in some cases, at least within a few years. My second point is in regard to the number of cases in our midst which have entered through Ellis Island. I think all who have a large foreign population to deal with appreciate this fact. If you doubt what has been said in regard to the superficial character of the examination at Ellis Island, you can be very much enlightened by spending a day there. It is practically impossible to diagnose these cases among the immigrants who come by thousands, as they pass in the line. A great many will necessarily slip through. But if congressmen can be interested in the Dillingham bill, a large number of these cases of trachoma can be shut out. This bill provides practically for the illiteracy test, and requires that the immigrant shall be able to read a certain number of sentences in his own language. I commend this bill to your careful study. It strikes at the root of this trouble. You and I see the effects of this disease and we appreciate them. The majority of the people do not. Your congressman does not, but if each of you will appeal to your congressman and show him the effects of this disease, then this discussion will have point and will have produced some results.

DR. M. WIENER, St. Louis: At the Oklahoma state meeting a few weeks ago I heard a great deal of discussion among the ophthalmologists as to the method by which the examination for trachoma was conducted. I am not speaking from experience, but I should like to ask Dr. Kerr and Dr. McMullen what are the preparation and the qualifications of the examiners who reported on these cases of trachoma. The ophthalmologists in Oklahoma held that there were a great many cases called trachoma which were not trachoma. The health authorities of St. Louis have requested the ophthalmologists to report trachoma and ophthalmia neonatorum.

DR. EDWARD JACKSON, Denver: A trachoma investigation was made of the 225 inmates of the State Home for Dependent Children, in Denver. A physician who has done a large amount of work examining schoolchildren reported that, on examination, over a hundred had trachoma. Those that were diagnosed as having trachoma were sent down to the clinic and examined by Dr. Coover, who found thirty cases. A single examination for trachoma can only show the acute and severe cases. I suppose that those in charge of the inspections at Ellis Island and elsewhere only feel justified in excluding cases that are more or less pronounced. Cases are constantly coming in that can hardly, with justice, be excluded, and yet may develop acute exacerbations and become centers of infection. I agree with Dr. Zentmayer that the work at the immigrant stations is being done extremely well and has accomplished much good. When I was serving at Wills Hospital twenty years ago, nearly all our cases of acute trachoma were in immigrants, of two or three months' standing. They had apparently been infected during the passage. But it only

V. 6
1913

furnishes a starting-point for a campaign against trachoma. A year ago I talked with the chief medical officer of Porto Rico. Trachoma was one of his most serious problems, although there was very little trachoma in Porto Rico. It was not serious because immigrants from other countries brought it in, because he could easily manage them, as they are managed at the immigrant stations in the United States, but it was serious because citizens of the United States were continually coming from the United States and bringing trachoma with them. There has been considerable agitation in Austria-Hungary against the return of those who have been in America for several years, and have become infected with trachoma, and are bringing it back into that region and increasing the amount of trachoma, according to the ophthalmologists. The same agitation has been started in certain parts of Italy on the same account and it is proposed to impose restrictions on such return. The question has certainly reached the stage in this country at which the suggestions touched on by Dr. Lewis are proper—to recognize every case of actual trachoma in the community and treat it as a contagious disease, and to adopt measures that will relieve us from such centers as those on the Indian reservations and in the mountains of Kentucky, and in parts of Missouri, Arkansas and Oklahoma among the white population. That means a campaign for legislation in several states for placing trachoma among the recognized contagious diseases that are a serious menace to the public health.

DR. FRANK ALLPORT, Chicago: It has been suggested that a fund be set aside by the legislature for the relief of trachoma, for instance, in Kentucky. We have some wealthy states in this country where money can be set aside—New York, Illinois, Pennsylvania and various other states—but without any disrespect to Dr. Stucky I may say, and I think he will endorse it, that Kentucky is a poor state, and even this pathetic and heart-rending lecture might be delivered to the legislature of Kentucky without any financial results whatever. Not that they would not be willing, but they have not the means, and there are many states in the South and Southwest where an appeal for funds would be useless for the same reason. We will have to raise money in other ways, either by national legislation or charitable contributions, or by funds furnished by some such organization as the Russell Sage Foundation. Dr. Fox's statement with regard to the curability of trachoma has been made, I believe, with due deliberation and experience. I have seen dozens of cases under Dr. Fox's care at the Medico-Chirurgical that have been cured. I do not believe Dr. Fox would say that all cases can be cured, but most of them, either with thorough grattage, such as we all perform, or with removal of the cartilage. Of course the effects such as rupture, staphyloma, etc., are not going to be cured, but I feel that the vast majority of these cases can be cured so that they are placed on the non-contagious side. I speak not only from observation in the clinics, but from my own observation and experience, which of course is limited, because we do not get a great number of such cases in Chicago.

DR. J. S. LICHTENBERG, Kansas City, Mo.: In explanation of the fact that some of these immigrants get by the inspectors at the ports of entry, all of us who have been abroad have seen numbers of these patients in the various clinics treating for trachoma with the intention of coming into the United States. At Moorfields, there is a special room for trachoma, and most of the patients, I was told when there, expected to become immigrants. They are apparently cured. The evidence of the disease is so slight that it cannot be detected. They then come into this country and have recurrences and become foci of infection.

MR. HENRY COPLEY GREENE, Boston: In our experience in Massachusetts we do not find a great deal of blindness due to trachoma. At the same time a certain number of cases of trachoma do slip through in spite of careful inspection at the ports. If an immigrant at the time of landing is liable to become a public charge within three years, he may be deported, and for the preservation of our own citizens it seems very important that the law should be enforced. During the last administration there was a certain amount of what I call sentimentalism about the deporting of immigrants. In these cases with low vision the people do not all go to Dr. Fox or some equally eminent practitioner. They slip in, their eyes are neglected for considerable periods, and they become, not totally blind, but so much disabled that they cannot earn a living. I think that oculists can help the immigration authorities and help in the protection of the community by seeing to it that these patients, who, at the time of landing are liable to become public charges, are deported.

DR. JOHN MCMULLEN, Washington, D. C.: It is unfortunate that we have to rely absolutely on the clinical aspects for the diagnosis of trachoma, and it is hoped that the cause of this disease will soon be discovered. The medical officers of the United States Public Health Service see, among immigrants, a large number of cases in all stages of the disease with its various sequelae. An immigrant may be deported within three years after his arrival, instead of two as has been stated, if found to be suffering from trachoma. I have seen Dr. Fox do his grattage in one case and, while of course grattage is not new, he apparently performs a very radical operation. I have no knowledge of this radical treatment except what Dr. Fox tells me. I do not wish to convey the idea that I believe trachoma to be incurable, but I know of no treatment, medical or surgical, which will surely give a quick cure in this disease. There are many thousands of trachomatous aliens in Europe and it would not be practicable to permit them to come to this country and be cured here. I agree that trachoma is a dangerous, communicable disease and it should be reported. Several thousand immigrants arrive daily at Ellis Island and the doctor on line inspection passes only those eyes which are plainly normal. He does not attempt to make a diagnosis, but puts aside any immigrant presenting any abnormal appearance of the eyes and the case is carefully examined in a room reserved for this purpose. If the case is at all suspicious of trachoma or a conjunctivitis of any degree is present, it is sent to the hospital and given observation care and treatment for days or perhaps weeks. The diagnosis of trachoma in an immigrant is made only after observation and treatment in the hospital. Any case of conjunctivitis that can be cured in a few days or weeks is not certified as trachoma.

DR. J. W. KERR, Washington, D. C.: At the last meeting of the American Medical Association a resolution was adopted calling on the Public Health Service for an investigation of trachoma in the United States. It should include a determination of the prevalence of the disease among ninety millions of people, but for the present this can be done only here and there. The passing of such a resolution is an evidence of the shortcomings of our system of reporting morbidity, which will have to be improved as one of the first steps to be taken in meeting the trachoma problem. We must have uniform state laws, because the government cannot force the states to report diseases. Nevertheless, in accordance with the resolution mentioned and also instructions from Congress, we have undertaken to make studies of the prevalance of trachoma. You are already familiar with the Indian investigations. In addition, we have examined several thousand schoolchildren in California, and over fifty-two thousand people in Minnesota within the last year. Approximately forty-eight thousand of this latter number were persons other than Indians, and Dr. Clark found only seventy-seven cases of trachoma among them. This total number includes about thirteen hundred miners in St. Louis and Itasca counties, and the undue prevalence of trachoma among them brought up the general percentage for the state. This is mentioned especially to emphasize the fact that we are probably not facing an epidemic. We probably have about as much trachoma now as we have had for a score of years, only we did not know of its prevalence then. The responsibility for the reporting and care of trachoma cases rests with the states, with the assistance of the government. Not only official agencies must be utilized, but private agencies and the great body of the medical profession. We have found difficulty in making inspections in two large cities in this country. In one we

were refused the opportunity to examine the schoolchildren, and in the other requests for the making of such examinations were ignored. If we are to ascertain conditions we must have the aid of state and local public health officials, which we now have, and we must have the aid of the medical profession and the boards of education. Dr. Lewis spoke of there being 2 per cent. of trachoma among the Indians of New York. Our results showed that it was 0.2 per cent among 943 Indians examined. Referring to Dr. Wiener's request for information as to the experience of our officers with trachoma, I think the discussion he heard in Oklahoma related to officers of the Indian Service. We heard the same criticism of the officers of that service. The disease, however, is highly prevalent there. We sent one of our most experienced officers to Oklahoma, one who had been at the Ellis Island Hospital for over six years and who has seen much trachoma. We did not send an officer into the field who had not had years of experience in trachoma, with the possible exception of one, who had had experience but had been out of the work for a while. He was sent to a state where there were practically no Indians present. The officer who reported on this work in Minnesota has been engaged in trachoma work for over ten years. Similarly, those sent to the Dakotas, Washington, California and other states had long experience. Our officers do not pay special attention to other diseases of the eye, to which you devote your lives, but they do know trachoma.

DR. J. A. STUCKY, Lexington, Ky.: I am sure that trachoma is curable. I hope that when we establish a clinic in the mountains of Kentucky, Dr. Fox will come down and stay a week or two and show us the grattage procedure, and then tell us what to do with the sequelae of the disease. I reported these conditions to the Kentucky State Board of Health two years ago, but to no purpose. The State Board of Health has a mere pittance of money. The state of Kentucky is wealthy in everything but money. We are over two and a half million dollars in debt. The State Board two years ago appropriated $100 for this work, which would pay part of the expense of one of my trips. It has not been paid yet. Some one said, "Why don't you go to the legislature?" I asked for that privilege, and I was told by the "powers that be" that it would defeat the efforts then being made toward getting more money for the State Board of Health. We appealed to the government and it answered promptly. Shall we appeal to the masses? My own state pride rebels at the idea of asking other states to help us, but if we can not do it ourselves that is all there remains for us to do. It is up to the medical profession to assist in educating the people who are blessed with more than their share of this world's goods to help save the last remnant of the real American, the people who came with Daniel Boone over these mountains a century ago.

TEMPERATURE OF THE CONJUNCTIVA

LUCIEN HOWE, M.D.
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I. EARLIER METHODS OF MEASUREMENT

Other students, appreciating the probable importance of this subject, have attempted similar measurements, but all of them were made with some modification of the mercurial thermometer. One form of this which attracted considerable attention was suggested by Galezowski. It had a flat bulb small enough to pass easily beneath the lid, and the tube extended 8 or 10 cm. at right angles to the bulb; but the glass of the bulb was so thin that the least pressure of the lid produced a rise of the mercury, and of course constant readings were impossible.

II. THE THERMO-COUPLE

The thermo-couple is also called the "thermo-electric junction," or "thermal junction."

If two strips of metal of unequal conducting power are soldered together, the line where they meet is of course a "junction." If the free ends of these two strips are connected, and then if the line of their first junction is heated or cooled, that junction becomes a "thermo-couple," or thermo-electric junction. An electric current is then generated throughout the circuit, and if a galvanometer be placed in that circuit the strength of the current will be indicated by the index.

Or again, we may arrange two such couples in the circuit, placing the couples so that they are technically in "multiple" or "parallel," having the galvanometer in series with one of the connections. Such an arrangement is sketched in Figure 1. Then if both couples are heated or cooled to the same degree at the same time, the electric current developed by each couple neutralizes, as it were, the current which is developed in the other and no change is registered by the galvanometer.

If one of these couples, however, is heated or cooled more than the other, the excess of electric current developed will be shown by the index of the galvanometer. This arrangement of two thermo-couples has been used for some years in laboratories and certain industrial establishments for measuring slight differences of temperature between two mediums.

When my attention was first called to the great sensitiveness of this arrangement, it seemed rather odd that no ophthalmologist had used it to measure the temperature of the eye. The reason, however, soon became

Fig. 1.—General plan of the arrangement of two thermo-couples with a galvanometer in circuit.

evident; for though the principle was well known and simple enough, its application was by no means easy.

These difficulties, however, have apparently been overcome, and the following is the general plan of the two thermo-couples which have proved most useful for measuring the temperature of the conjunctiva as compared with that of the mouth:

1. A very fine covered copper wire extends about 8 or 10 feet from one post of a galvanometer.

2. The free end of this copper wire is fused to one end of a composition wire, which is particularly useful for delicate thermo-couples. Several kinds of such wire are on the market. The one which I used is known as Ideal No. 32. The point of fusion of these two delicate wires forms the first of the two thermo-couples.

3. The other end of this Ideal wire is fused with a second piece of copper wire. This junction forms the second thermo-couple.

4. The other end of this copper wire extends about 8 or 10 feet to the second post of the galvanometer.

Theoretically these two couples would be sufficient to indicate on a suitable galvanometer any difference of temperature between them. But practically we know that:

A. The junction of the copper, and of the composition wire must be secure.

B. The two wires must be thoroughly insulated, and not easily injured.

C. They must be so protected as to be easily and thoroughly disinfected.

D. They must "balance" exactly. That is, neither should produce any current in excess of its fellow, when both couples are at the same temperature.

It is desirable, therefore, to glance at the special forms of the thermo-couples which, after numerous trials, have been found most suited for introduction into the conjunctival sac and into the mouth, respectively.

A. The couple for the conjunctiva. This is seen, natural size, in Figure 2. In making records it is convenient to designate this one as "Couple A," and to attach its wire to the post of the galvanometer which corresponds to black numbers on the scale.

B. The couple for the mouth. This is seen, natural size, in Figure 3. In making records it is convenient to designate this one as "Couple B," and to attach its wire to the post of the galvanometer which corresponds to red numbers on the scale.

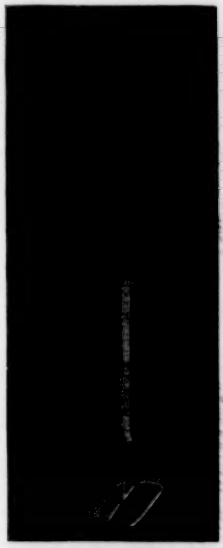

Fig. 2.

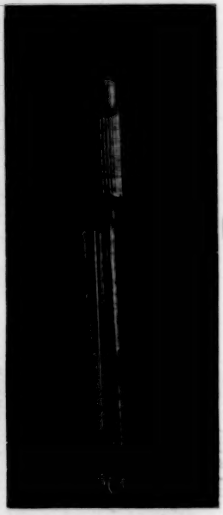

Fig. 3.

Fig. 2.—Thermo-couple for conjunctival sac (actual size).
Fig. 3.—Thermo-couple for mouth (actual size).

III. THE GALVANOMETER

Although any variety may be used, the galvanometer found most convenient for this purpose was the D'Arsonval, Type P, mounted on a tripod. This is seen in Figure 4. Descriptions of the D'Arsonval galvanometer can be found in any treatise on magnetism. Any unequal heating or cooling of the two couples produces a current which causes a suspended mirror to turn from side to side. This mirror with its suspension is seen enlarged in Figure 5. The mirror is viewed through a telescope, and reflects the figures of a scale. This scale on the horizontal strip is 50 cm. long, graduated in millimeters, and numbered 25 red to 0 to 25 black. As this scale is on the side of the strip facing the galvanometer, of course it is not visible in Figure 4.

IV. SENSITIVENESS OF THE THERMO-COUPLE

According to measurements made at the factory in Philadelphia with my couples, 1 mm. of the scale corresponds to about six hundredths of one degree Centigrade (0.06 C). Practically the same results were found here. Now it is not difficult for an observer to distinguish a movement of the index of a half or even a quarter of a millimeter. That is to say, the couples arranged in this way show a difference of temperature of 0.03°C. or even 0.015°C. This is the sensitiveness expressed in figures.

An illustration shows better what that means. If the hand is placed 3 or 4 inches from one of these couples, the radiated heat is sufficient to make a very distinct movement of the index of the galvanometer. If the finger touches one couple the index at once swings over 30, 40 or even more millimeters. Or if one couple is held in the hand and gently breathed on, the index flies rapidly not only to the end of the scale, but so far beyond as to make any reading impossible. In other words, we have here a method of measurement far more sensitive than by any thermometer which could possibly be used.

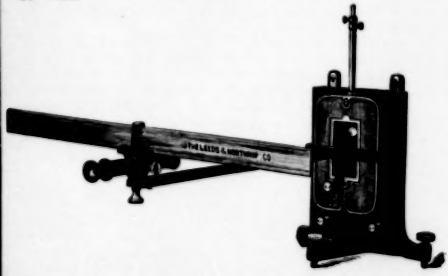

Fig. 4.—D'Arsonval galvanometer for registering variations of temperature.

V. METHOD OF MAKING THE MEASUREMENTS CLINICALLY

As a preliminary we must make sure that the two couples "balance" each other; in other words, that the galvanometer registers zero when the two couples are in some medium — air or water — in which their temperatures are exactly the same. It is also essential to know how many seconds or minutes are necessary for such a balancing to take place, as this depends largely on the relative thickness of the glass which insulates each thermo-couple, and somewhat on the distance which each is covered. Let us suppose that with a given pair of couples we have ascertained that such a balancing takes place after two minutes and a quarter. Also suppose that we have applied a 5 per cent. solution of cocain two or three times to the conjunctiva to be examined. The patient being then seated comfortably and conveniently near to the galvanometer, Couple B is placed in his mouth. He is asked to close his lips on it, and allow the glass tube to rest against the side of the tongue, as is done when we use a clinical thermometer. This couple must of course first remain in that position for at least the two minutes and a half which had been found necessary by previous experiment to bring it up

to the body temperature. Having done this, the surgeon takes Couple A with the thumb and index-finger of the right hand, as indicated in Figure 6, the lid is raised, and the couple introduced gently beneath the lid.

Care should be taken to have the portion which is beneath the lid actually in contact with the globe. If the point is bent forward so as to cause the lid to project and produce an air space between the lid and globe even a few millimeters wide, the effect is seen at once.

While the two couples are thus held in position by the surgeon, an assistant looks through the telescope of the galvanometer to observe the changes in the index, and to record them as they occur. At first the index may show that the mouth registers as much as 1 or 2 degrees above the temperature of the eye. But if care is taken to have the patient keep Couple B gently but constantly against the tongue, and if the operator is equally careful to rest Couple A against the eye, after from three to four or five minutes the temperature becomes gradually more equalized, until, under normal conditions, Couple A, in the culdesac near the inner or outer canthus of the eye, registers from 0.2 to 0.3° C. lower temperature than that of the mouth.

Fig. 5.—Mirror of the D'Arsonval galvanometer, showing its upper suspension, and attachment below to a coil of fine wire.

VI. GRAPHIC REPRESENTATIONS OF VARIATIONS IN TEMPERATURE

This can be shown on a system of coordinates in which the zero line represents a perfect balance between the two couples, A and B, no matter what the absolute temperature may be. Each division of the abscissa (the vertical line) below the horizontal zero line shows that the index on the scale of the galvanometer has moved 2 mm. toward the black side—in other words, that the temperature of Couple A (the one used for the eye) is 0.12 degree Centigrade warmer than Couple B.

On the other hand, each division of the vertical line above the horizontal zero line shows that the index of the galvanometer has moved 2 mm. toward the red side of the scale—in other words, that Couple B (in the mouth) is 0.12 degree warmer than Couple A.

Each division of the horizontal lines represents one-fifth of a minute. Such a graphic representation of the variation of temperature in one observation is shown in Figure 7.

VII. RESULTS OF THESE MEASUREMENTS

After this necessarily detailed description of the construction and method of using these thermo-couples, the question naturally arises, What do we learn from them? The results may be briefly stated as follows:

1. We know now and for the first time that the temperature of the culdesac of the conjunctiva near the outer or inner canthus is usually from about 0.3 to 0.4 degree C. lower than that of the mouth. This finding was at first rather surprising, and was naturally attributed to some fault in the instrument or in manipulation. Repeated trials, however, on different individuals seem to establish the fact beyond question. The reason of this is probably that the couple placed in the mouth rests against the tongue, and is protected by that on one side, and the thick layer of facial muscles on the other. On the other hand, the globe of the eye is protected only by the lids, much thinner of course than the cheek, and therefore not only colder, but more sensitive to variations of external temperature.

2. The temperature of the conjunctiva immediately over the cornea in six individuals was found to be on the average 0.1 degree C. lower than at the outer or inner canthus.

It will be observed that 36.9 C., or about 98.6 F., can be considered as the normal temperature. In several of the measurements the patients held in the mouth not only Couple B but also a Centigrade thermometer graduated in fifths of a degree, and accurately corrected by

Fig. 6.—Method of using the two thermo-couples.

the Bureau of Standards. This gave the absolute temperature of the mouth, from which by comparison it was possible to obtain the absolute temperature of the conjunctiva. In these measurements also care was taken that no cold or warm fluids or food had been taken into the mouth for some time previously, as such substances affect the temperature for minutes or even for a few hours.

3. It is possible by this method to measure for the first time to what extent cold or hot applications to the outer portions of the lids really change the temperature of the conjunctiva. It was found, for example, in one case that when pieces of cotton moistened with ice-water were applied to the lids in rapid succession four or five min-

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utes, the temperature of the conjunctiva could easily be reduced 1 or about 1.5 degrees Centigrade.

On the other hand, the difficulty in reducing the temperature below that increased rapidly with each fraction of a degree.

This point is of importance in connection with the treatment of bacterial infections. It is well known that many of the forms which invade the conjunctiva have their vitality lessened in proportion to the decrease in temperature. Practically, however, it is difficult or impossible to produce such a change in the temperature as prevents the growth of these bacteria in the test-tube. This is a phase of the subject which seems worthy of much more careful study concerning each separate germ.

4. It is probable that the thermo-couples here described will prove of some value in at least two other directions. These are:

A. Indicating changes of temperature which accompany certain inflammatory conditions of the cornea, uvea or other portions of the globe itself.

B. Inasmuch as efforts of accommodation and convergence are often followed by slight injection of the conjunctiva, it is quite possible that with these two couples we may be able later to obtain a better idea of

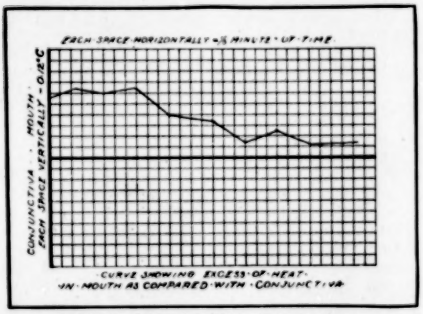
EACH SPACE HORIZONTALLY = ½ MINUTE OF TIME
CONJUNCTIVA - MOUTH - 0.5°C
EACH SPACE VERTICALLY = 0.1°C
CURVE SHOWING EXCESS OF HEAT IN MOUTH AS COMPARED WITH CONJUNCTIVA

Fig. 7.—Chart of variation of temperature.

the metabolic changes which take place in that complicated process which we call eye-strain.

In a word, the field which thus opens before us for the first time seems to be one ripe and ready for a harvest of interesting facts by the ophthalmologists of the future.

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ABSTRACT OF DISCUSSION

DR. EDWARD JACKSON, Denver: A paper of this kind suggests to me certain possible applications; and also a question or two which may be of practical importance. It is impossible that such an apparatus should come into clinical use until it has been worked out into a somewhat permanent form. It should be standardized so that it can be managed by different men, eliminating any serious personal equation, so that the results will be fairly constant. Then a considerable amount of laboratory work would have to be done with it to determine facts to be observed in connection with disease. Dr. Howe has mentioned the point that first suggests itself in the therapeutic application. In the reduction of the temperature of the eye it seems likely that our therapeutic measures cannot be extended greatly. But with some accurate means of determining what we accomplish by it, heat could be used considerably more freely, and more effectively than it has ever been used. It was pointed out a great many years ago that the eye tolerates very high temperatures on the surface. Dr. Lippincott of Pittsburgh recommended for corneal ulcers raising water to the boiling point and dropping it as quickly as possible on the ulcer. So if we knew just what we were doing, and what could be done, heat could be applied more effectively than ever before in ocular therapeutics. The question arises as to whether the mouth is the best point of comparison. In the beginning it may be well to compare the temperature of the eye with that of the mouth. I can see the value of experiments continued along that line, but it seems to me that when it comes to be applied as a clinical method it would be better to compare the temperature of the two eyes? What reason is there for thinking that that would not be a better line of investigation?

DR. MELVILLE BLACK, Denver: There are two or three practical points in connection with this demonstration, and one is, I think, in regard to how long it is necessary to make an application of heat or cold to either raise or lower the temperature of the part and have it maintained. For instance, we have been in the habit of making hot applications for about five minutes. We believe that after that time the hot applications cease to be of benefit and are detrimental. This procedure ought to explain why. It may be that after the application of heat continuously, for a certain period of time, we would find, by this apparatus, that it does not change the temperature of the conjunctival sac, and that possibly is also true with regard to cold. We know that we can apply cold for a considerable time without detriment and it would be interesting to know what thermal changes there are in the sac after one or two minutes. Will Dr. Howe explain more fully how he made the eyes cold? He speaks of making ice applications. There are many different ways of making ice applications. Has he used the ice-bag with pulverized ice, or ice pledgets of gauze changed every half-minute, and if so how much reduction of temperature was occasioned by this? In Dr. Howe's paper I have failed to get satisfactory answers to these questions.

DR. ALLEN GREENWOOD, Boston: I wish to call the attention of the members to the experiments of McGillivray of Scotland, who, though not using a delicate instrument like this, tested the temperature of the conjunctiva and cornea and found that they were several degrees Fahrenheit lower than the ordinary temperature. With the lids closed the temperature was raised one or two degrees and with the lids open was lowered the same amount. From this, and from the fact that bacteria grow slowly when the temperature is reduced, he worked out a theory of testing corneal conditions by such means as allowed the patient to keep the eyes open most of the time, and he proved conclusively to himself that the lowering of the temperature brought about by the exposure of the cornea to the air inhibited the growth of germs in ulcer of the cornea. This is along the line of what Dr. Howe has been doing so skilfully and scientifically.

DR. MARK J. SCHOENBERG, New York: There is a possible source of error in the fact that Dr. Howe has used 5 per cent. cocain in the conjunctiva, which is vasoconstrictor, and this might reduce the local temperature. He found the temperature about 0.4 degree lower on the conjunctiva than in the mouth. If Dr. Howe had used a solution of cocain in the mouth at the same time he used it in the eye, then the control, in my mind, would have been better. I would suggest the use of holocain, which has no vasoconstrictor action. By studying the intra-ocular temperature of normal animals and in eyes which have been subjected to various injuries and pathologic conditions, something could be learned of the temperature of the interior of the eye in certain pathologic conditions. Dr. Jackson justly called attention to the point that the temperature should be compared in both eyes at the same time. In tuberculosis of the lung it is found that the temperature of the axilla is higher on the diseased side. Even the temperature of the skin has been measured by special thermometers and has been shown to be a little higher on the side of the tuberculous lung. The same thing could be made out with the temperature of the conjunctiva.

measured in both eyes, as to whether pathologic changes were going on in one eye.

DR. WILLIAM H. WILDER, Chicago: We are all acquainted with the clinical fact that hot applications help certain deep-seated inflammations of the eyeball. The supposition is that with the Bier treatment we actually raise the temperature of the part to which the heat is applied, thereby quickening the circulation and bringing fresh blood and antibodies to combat the infectious elements. It would be an extremely interesting practical point to know if we are using hot applications in a way to increase the temperature so as to produce increased circulation through the deeper structures of the eye. I believe these experiments may be able to elucidate some of these points.

DR. GEORGE H. PRICE, Nashville: It is a well-known fact that all the surfaces of the body and internal structures differ in temperature; the eye, ear and nose show variations. Dr. Howe's ingenious instrument is for measuring the normal temperature of the conjunctiva and for detecting variations due to disease. Living substances will stand very high and very low temperatures. A fish may be frozen up in ice, thawed out, and live, but boiling kills it. Cold only retards inflammatory processes due to germs. If we reduce the temperature until the germ is killed, the tissue is also killed. Sufficient heat will destroy any germ which produces coagulation. Cold retards germ production, but heat encourages it up to a certain point, beyond which it stops it. By this instrument we may be able to fix the limits for best results in treatment. If reproduction of the germ about the eye can be held in abeyance the disease will be checked. In so-called non-infectious conditions of the eye it might do some good, giving us an indication as to the temperatures best suited for combating such conditions.

DR. LUCIEN HOWE, Buffalo: In this first paper it has only been possible to present a description of the apparatus and give a few suggestions as to its application. But the problems which have been suggested here, and one or two others which might be worked out with these thermo-couples, have been already considered and some of the preliminary work has been commenced. It may be one or two years before it is safe to formulate any definite conclusions. For example, the temperature of the interior of the globe has been measured in two cases in which a practically normal eye was about to be enucleated on account of malignant growths in the orbit. These measurements seem to show that the temperature of the interior of the globe is a little higher than that of the conjunctiva and lower than that of the mouth. It may also be stated that measurements have been made of the degree to which it is possible to lower the temperature of the conjunctiva by applying ice in a certain way or how the temperature can be raised by applying heat. This last is apparently of great importance with regard to the life of the Neisser gonococcus, because that germ can be killed, at least in the test-tube, by a temperature only slightly above the normal temperature of the conjunctiva. Now, for the first time, we can measure just what temperature is produced by this or that method and thus we can control our treatment intelligently.

BLEPHAROCHALASIS

REPORT OF TWO CASES WITH THE MICROSCOPIC EXAMINATION *

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NEW YORK CITY

Blepharochalasis is the name suggested by Fuchs in 1896 for that condition affecting the skin and the subcutaneous connective tissue of the lids, with atrophy.

This condition has been reported repeatedly since then, but still must be considered as a comparatively rare affection and up to the present I have not been able to find any cases reported except those of the foreign clinics. The disease is not a new condition, as McKenzie[1] as early as 1854 called attention to a disease of the lids which he thought was a ptosis with hypertrophy following edema, and which was later followed by a relaxation and drooping of the skin of the lids. In nearly every instance in which the affection has been reported it has been given a new name by the individual who reported the case, and this has been most unfortunate, as it has only added confusion in our study of the disease. Perhaps if there was a clearer understanding of the etiology and the pathology of the cases seen and reported there would not be all this difference of opinion as to its proper nomenclature.

It seems wise to classify it with the dystrophies of the skin and Fuchs has included in this class ptosis adiposa and elephantiasis. Von Ammon spoke of this condition of the lids and called it epiblepharon, ptosis adiposa or ptosis atonica. The latter name fairly well describes the condition of the lids in the later stage of the disease.

Sichel[2] in describing a case enumerated nearly all of the symptoms, but he regarded it as a true ptosis of the lids, which as we now know, from our further observations, is not the case. Arlt,[3] reporting his case, called attention to the rosy appearance of the skin, but apparently overlooked the marked thinning and atrophy. Schwalbe was the first to use the term "blepharochalasis," but he used it to describe the results of his operation on the lids of patients who had shrinking and atrophy of the conjunctiva and the subcutaneous connective tissues of the lids after trachoma. His operation was designed to bring about a relaxation of these tissues and he called the procedure by the name just mentioned.

Many other names have been suggested from time to time by the different authors and some of these will be only mentioned; the wisdom of their selection will be left to the reader. Panas called it "ptosis cutanea"; Terson, "dermatolysis palpébrale," which was the name suggested by Dr. Crary, who examined my first case for me. Frankel designated it as "paupières en besace," Dutoit "pseudoleukemia lymphatica," and Rohmer, who has reported four cases, thinks that "angiomégalie des paupières" best describes the condition.

"Blepharochalasis" is the best name that has been suggested. Rejecting the idea of ptosis and admitting the existence of edema, with sometimes a consequent stretching, thinning and atony of the skin and subcutaneous tissues of the lids, this term includes all of the most important changes.

In regard to the etiology of this disease, nationality does not seem to be a factor, although in all of the cases that have been reported so far, except my second case, the patients have been of foreign birth. This condition has not been reported as occurring in the colored race, except in Shoemaker's case, which was not reported as blepharochalasis, and therefore must be considered only as being possibly of such origin. With the exception, perhaps, of three, all of the cases of the blepharochalasis reported have been in girls. The youngest patient on record was 8 years of age and the average age 10 to 14. It seems to bear some relation to the beginning of menstruation and, from study of the records of the cases so far reported, I find that nearly all of them began at or near the time of the

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

1. MacKenzie: Practical Treatise on the Diseases of the Eye; Ed. 4, London, 1854, p. 187.
2. Sichel: Ann. d'ocul., 1844, xii, 188.
3. Arlt: Handbuch für Augenheilkunde (Graefe-Saemisch), iii, 454.

first period. In my second case reported in this paper there was a history of severe sunburn having preceded the appearance of the trouble, but I do not think that it had anything to do with the appearance of the trouble.

There are several cases in which the lesion appeared after severe physical exertion (Fuchs), but this in itself would not seem to be sufficient to explain all of the signs and symptoms of the disease. His case was that of a man who noticed the curious swelling of the lids after blowing a horn. A history of a profound mental shock or fright has preceded the appearance of the swelling, and in some of the cases it has been first noted after a long spell of crying. This all emphasizes the importance of carefuliv considering the emotional and psychic origin of this disease.

Recurrent attacks of angioneurotic edema have been given by Fuchs[4] and Rohmer as the cause. This seems quite a possible explanation for some of the cases, considering the age of the majority of those reported, together with the sex and history of repeated attacks of the edema and the consequent deformity.

Errors of refraction have not been considered of much or any importance as a cause, and in neither of my cases were they of such a nature as to make me regard them as a possible factor. Schmidt-Rimpler[5] thinks that it can best be explained by a hernia of the orbital fat. It has been suggested that it is directly due to changes in the vascular and lymphatic systems. But there has never been found in any of the cases any special involvement of the general lymphatic system. The change noted in the vessels has been an increase in size but no increase in the number of vessels in the part affected.

As far as my own observation has gone it appears that blepharochalasis begins with an edema of the subcutaneous connective tissues of the upper lid between the skin of the lid and the tarsal plate; the presence of this edema affecting, after a time, the elasticity of the tissues involved, with a consequent atrophy of the skin and subcutaneous tissues of the lid. I would like to call attention again to the importance of the time of life at which it appears and its close association with the beginning of menstruation, which I believe is a causal factor. Just why it affects the upper lid and remains confined to that portion of the orbital tissues I am unable to explain. There may be found in our further studies of this disease that autointoxication plays some part in the etiology of some of the cases of blepharochalasis. Inheritance has not been of great importance in the causation of this condition. In only two of the cases reported is there an indefinite history of the same condition having existed in one of the parents.

The microscopic examination of the tissues removed from the right lid of my second patient was of sufficient interest to report in detail.

Skin section was stained with hematoxylin and eosin after hardening of the tissue in the usual manner. The epidermal layer of cells was of normal thickness and there were no special changes noted in any of the different layers. This section of skin was compared with a piece of skin that had been removed from a normal eyelid, and the changes in the rete malpighii that have been spoken of by several observers were not found. There was a slight round-cell invasion noted in some portion of the corium and a widening of the spaces in the subcutaneous tissue of the corium. This widening and stretching of this portion of the corium was probably the cause of the blepharochalasis, or it may also have been the result of this disease. This condition of the corium gives the appearance of edema as described in these cases, but it does not show any fat formation in this portion of the lid. Special staining by the Weigert method for elastic fibers did not show any increase of this special tissue in the section. Some have asserted that the elastic tissue is increased and that there is an increase in the blood-vessels and lymphatics, but neither of these changes was found in my sections. Sections of the subcellular connective tissue showed the blood-vessels well filled with corpuscles and dilated; but no increase in the number of vessels and nothing of any importance was found, except the dilatation of the subcellular spaces.

The fatty mass which was removed from the under part of the superior portion of the orbit seemed to be enclosed in a capsule and had all the appearance of a fatty tumor which shows a great increase of blood-vessels all filled with blood. A considerable number of fat-cells and stroma with a well-marked connective tissue stroma made up the main part of the tumor. The decided increase of the blood-vessels in this tissue suggests that it was not the normal orbital fat.

The lacrimal glands which were removed from both sides were examined and the glandular portion was found to be healthy, and at places the capsule was seen. There was no special engorgement of the vessels, but there were several areas showing some round-cell invasion. This, however, was not marked and may have been due to the inflammatory reaction in this portion of the orbit. These areas of round cells consisted of leukocytes and were in the immediate vicinity of the blood-vessels.

It may be well to say that I removed the skin and the subcutaneous connective tissue separately, which of course is to be regretted as it was not possible to study the relation as closely as I had desired. The lacrimal gland was removed separately from the fatty mass which seemed to come from the superior wall of the orbit and behind the septum orbitale.

There has been considerable opportunity afforded for anatomic and pathologic investigation of this disease. Most of the patients have had one form or another of operation for removal of the affected tissues of the lid, thus permitting microscopic examination of the specimens obtained. It has not given us much information, however, because the findings have been so widely different in many of the reports. In most of the observations there has been an atrophy of the different layers of the skin, but more especially the rete Malpighii, the presence of fatty tissues, an increase in the size of the vessels, with a stretching and widening of the meshes of the connective tissue, is all that has been noted. No one seems to have given any explanation for the edema or the infiltrate which is found in the meshes of the subcutaneous connective tissue of the lids. It looked very much like a fatty infiltrate in my case and Bach[6] found, when operating on his patient, that there was what looked like a fatty tumor which seemed to come from beneath the upper orbital wall.

Fehr[7] describes very minutely what he found as far as the excised skin is concerned. He hardened the tissue in formaldehyd solution and stained it in various ways and at the same time treated some normal skin in like manner. The excised skin showed atrophy of all of the layers, but in the rete Malpighii layer he found almost complete destruction. The subcutaneous tissue was very lax and friable. The skin and the subcutaneous tissue were rich in blood-vessels, which were dilated and filled with normal blood-cells. In places there were pigment deposits which were probably due to the blood.

4. Fuchs: Ueber Blepharochalasis, Wien. klin. Wchnschr., Feb. 13, 1896, p. 109.
5. Schmidt-Rimpler: Centralbl. f. prakt. Augenh., 1899, xxiii, 297.
6. Bach: Arch. f. Augenh., liv, 73.
7. Fehr: Centralbl. f. prakt. Augenh., 1898, xxii, 74.

Part of the sections were stained dark-blue with orcein for elastic fibers, according to the Unna-Tanzer method, and the remainder of the tissue decolorized to a grayish red. The elastic fibers were still present in great numbers but much thinned and stretched, forming wider meshes than normal. The thick masses of elastic fibers found normally at the junction of the skin and the subcutaneous tissues were entirely absent. Schmidt-Rimpler says that he was not able to find anything characteristic in any of his examinations.

Rohmer[8] was able to examine the flaps removed from two of his patients. In Case 2 he found dilatation of the lymphatics and new-formed vessels, large sections of vessels (arteries and veins) engorged with blood in the skin and subcutaneous tissues. Not so much the number of the vessels as the caliber showed increase. There was a general thinning of the skin, but no study of the elastic fibers was made. In Case 3 the elastic fibers were carefully studied (Unna-Tanzer) and no changes were discernible. Inflammatory features appeared due to the cicatrix from a former operation five weeks previously. Arteries and veins were much increased in size but no changes were noted in the lymphatics. In closing he says that none of his studies has enabled him to form any conclusions that will throw light on the subject. Rosenstein[9] has demonstrated some microscopic preparations with marked atrophy of the elastic fibers, showing, besides a flattening of the epidermis and a tearing apart of the subcutaneous connective tissue masses, dilated veins and thinned walls. This dilatation of the subcutaneous connective tissue was noted by Fehr also and it is this portion of the lids which I think is the first to be involved in the disease. Whether this dilatation of the subcutaneous connective tissue is an edema or due to an invasion of fat is a question that, if once decided, will help a great deal toward clearing up the etiology of this disease.

There is no trouble in making a diagnosis of this condition after one has seen a case of blepharochalasis. In most of the cases reported it has occurred in young girls and is always limited to the upper lids; and it never gets well if left to itself, which is not the rule in angioneurotic edema. There are never any subjective symptoms, such as pain, heat or tenderness. It has been confounded with ptosis adiposa, but this should not occur if we remember that the latter disease is nearly always seen in the old, rarely or never in the young, and that it involves all of the lids, upper and lower, and is generally seen toward the inner half of lids. Many of the cases of "ptosis adiposa" reported have really been cases of this disease and Wooton has reported two cases of "ptosis adiposa" in young girls, on whom he has operated with good results. He now thinks they should have been diagnosed as blepharochalasis.

Shoemaker[10] reported a case of "bilateral enlargement of the lacrimal gland;" from the pictures of the case and the history, it would seem that this might also have been a case of blepharochalasis

Abstract of Case Report.—Miss M. G., colored, aged 19. At the age of 14, both upper lids became swollen, swelling more marked to the outer side of lids, ptosis of lacrimal glands, no change in the parotid or salivary glands, anemia present, no syphilis or tuberculosis. Condition not changed by treatment.

8. Rohmer: Arch. d'ophth., 1900, xx, 407.
9. Rosenstein: Centralbl. f. prakt. Augenh., 1902, xxvi, 233.
10. Shoemaker: Ann. of Ophth., 1904, xiii, 513.

Lambert[11] presented a case report of hypertrophy of the lacrimal glands, which may have been from the history and the microscopic findings, also a case of blepharochalasis.

Abstract of Case Report.—Miss X., aged 19. Condition appeared at the age of 13 with swelling of both of the upper lids, gradually increasing. Palpation revealed a tumor, which increased in size when patient leaned her head forward. Right side was larger than left. (This was true in both of my cases.)

Dr. Dixon's microscopic study revealed lacrimal gland and orbital fat. "Nothing observed as pointing to malignancy; therefore the diagnosis of hypertrophy of glands stands."

Hirshberg[12] reports a case of mumps der Thranendrüsen which has points in common with blepharochalasis.

Abstract of Case Report.—A girl of 15; swelling and redness of the upper lids; lacrimal glands palpable. Condition about the same over a period of years, in spite of treatment.

There are a number of conditions affecting the lids for which this disease might be mistaken.

Ptosis adiposa, as has already been said, presents some of the symptoms of blepharochalasis. There is a relaxation of the skin, but no true atrophy follows the condition no matter how long it may have existed. In ptosis adiposa the bagging of the upper lid is more marked at the inner side, and this condition is thought to be due to the relaxation of the bands of fascia connecting the skin with the tendons of the levator with the upper margin of the orbit. This condition has been referred to as "tear-bags" in the old.

Elephantiasis of the lids is seen in later life and often gives a history of having appeared after severe inflammation of the lids. It is frequently seen after attacks of erysipelas. In this disease the swelling of the lids may assume tremendous proportions. This swelling is a permanent thing and may hang down over the face. There is in some of the cases involvement of other parts of the body and it may be possible to demonstrate the presence of the *Filaria sanguinis hominis* in the blood. The skin is much denser and firmer than in the cases of blepharochalasis.

Lymphangioma of the lids is usually of the upper lids and in some cases may be limited to one side. The growth in the lids is rather firm and dense and there is often a form of blood dyscrasia which helps in the making of the diagnosis.

Plexiform neurofibroma of the lids is often seen in youth, but it has a rope-like feel to the touch, full of ganglionic nodes, may invade the temple, the lower lids and even extend into the orbit. It is usually unilateral.

Bilateral enlargement of the lacrimal gland simulates this condition very closely and might be easily mistaken for blepharochalasis. There is the slight bagging and sagging of the upper lid to the outer side, but there would not be the general fulness of the subcellular tissues and the thinning of the skin. Hirshberg thinks that bilateral enlargement of the lacrimal gland is not very rare and gives three causes for this enlargement: chronic inflammation following long-standing corneal inflammations; acute inflammation of the gland itself (this is very rare); subacute or nonsuppurative bilateral enlargement possibly due to paralysis of the third nerve.

Mikulicz' disease is quite a different condition and consists in the coincidental enlargement of the salivary

11. Lambert: Tr. Am. Ophth. Soc., 1900-1902, p. 403.
12. Hirshberg: Centralbl. f. prakt. Augenh., 1890, xiv, 77.

V. 6
1913

and lacrimal glands without pain and is chronic in its course. The disease is probably due to infection from the blood or by way of the ducts of the gland. In some of the cases of this disease there has been local disease affecting the conjunctiva or the cornea or a general disease was present, such as syphilis, tuberculosis, leukemia or menstrual disorders. As a rule it is not seen in the young but comes on in middle life. It might be possible to mistake such tumors as lipomas, adenomas, fibromas, affecting both lids, for blepharochalasis, but the bilateral appearance of such tumors is very rare.

CASE 1.—Miss F. V., aged 16, Bohemian. Family and personal history negative; no tendency to edema or swelling of any parts of the body. Usual diseases of childhood.

Present Trouble.—At 14 years of age, when menstruation began, patient first noticed the appearance of slight swelling of the lids. She states that at first the swelling would come and go, and later became permanent. She was treated without any improvement of the condition and came to the Manhattan Eye and Ear Hospital one year after trouble began.

Examination.—Patient was a fairly well-developed girl with a rather pale and anemic-looking skin, but good general health. She came on account of the deformity. There was a bagginess and drooping of the skin and subcellular tissues down over the edges of the palpebral margins. The skin was smooth and there was no tendency to wrinkling or folding, there was a

Fig. 1.—Case 1.—Condition after two years. Right side is most pronounced. Seton through the left upper lid.

pseudoptosis of the lids. The skin was slightly more pinkish over the area of swelling and the veins seemed to be slightly more prominent. This mass was easily compressible under the fingers and could be moved freely from side to side, giving the suggestion to the fingers of an edema. The lacrimal glands were in place and not enlarged. There was a marked wrinkling of the skin of the brow, from the constant effort on the part of the patient to elevate the lids, which were so much lower than usual on account of the weight of the mass. The lower lids were perfectly normal and the skin absolutely unaffected.

The Wassermann, von Pirquet and urine were all negative. The blood-count was normal as was also the hemoglobin. Search was made for the *Filaria sanguinis hominis* but without success.

This case was first regarded as one of elephantiasis. It was referred to Dr. Crary who made the following report: "The case is an unusual one and presents some difficulties. I believe the condition to be a form of dermatolysis, although in this condition there is usually a certain amount of hypertrophy of the skin, while in the case under discussion, it seems to me that there is a well-marked atrophy of the skin, associated with the laxity; but the term used is a broad one, and answers fairly well to fix the classification. I feel that the well-developed anemia, apparent from the color of the skin, pointing to a chlorosis, suggests a line of treatment and also suggests a possible neurotic condition, perhaps even a hysterical element."

Treatment and Results.—A seton of white silk thread was introduced into the upper lid of the left eye, entering near the center of the lid and this seton was carried under the skin of the lid and face, emerging on the face near the prominence of the maxilla (Figure 1). It was thought that the presence of the seton might set up irritation and cause absorption. This was allowed to remain for nearly two months without any appreciable change in the lid. Increasing doses of Fowler's solution were given over a period of a month without any effect one way or the other. Bland's pill and different forms of tonic treatment were given with no benefit. Roentgen ray and the high-frequency current were used for several weeks, but no changes were noted. Local use of collodion and pressure with adhesive-plaster straps and bandages at night did not alter the condition. The faradic current was used and the electric needle was inserted into the swelling; this was continued for two weeks or more but no great reaction followed the use of the needle and no contraction of cicatrix was noted. Patient became discouraged with the failure to get any improvement from the treatment and, as she refused operation, ceased coming to the clinic. On examination one year later the following changes were observed: The rounded swelling of the lids had disappeared and the skin hung down in a series of folds, with

Fig. 2.—Case 2.—Before operation Jan. 30, 1913. Most marked on the right side.

great relaxation of the skin and subcellular tissues of the lid. The skin looked much thinner and the surface of the skin showed the crackled cigarette-paper appearance described by Fuchs. The veins were more prominent than when last seen and the apparent ptosis of the lids was still present. It would seem that the lids will remain as they are unless some form of operative means is used to correct the deformity.

CASE 2.—Miss M. H., aged 14, white, American. Family and personal history negative. No members of the family have ever had angioneurotic edema.

Present Trouble.—Two and a half years ago, following a severe sunburn of the face and arms, patient's mother noticed a slight swelling and puffiness of the lids a few days after the blush of the sunburn had disappeared. The swelling has gradually increased and patient had seen a number of doctors but received no relief from any of the treatment. One of her doctors told her that it would go away of itself, evidently thinking that the condition was due to an angioneurosis. She came to the Manhattan Eye and Ear Hospital Dec. 20, 1912.

Examination.—Patient was a well-nourished girl, skin of good color, light hair, freckled and weighed 110 pounds.

Mother said that patient cried from very slight cause, was very sensitive and rather nervous and easily excited, but had never had any fits or convulsions. The skin of the upper eyelids was pinkish red, and hanging down in a baggy, pouch-like mass and partially covering the eyes. This swollen portion of the lids looked like an edema of the subcellular connective tissues. The swelling hung down over most of the palpebral edge of the lids and this was more marked at the outer commissure. There appeared to be a ptosis of the lids, which was more apparent than real and the sagging of the lid was due to the weight of the pendulous mass of skin and subcellular tissue. There was, however, a true ptosis of the lacrimal glands. The swelling of the subcellular tissues, the bagging of the skin and the ptosis of the lacrimal gland were much more pronounced on the right side. The superficial veins in the lids were more prominent and perhaps slightly engorged. There was no limitation of motion of the lid or eye. Emotional excitement and the menstrual periods seemed to increase the puffiness and the redness of the lids. The skin and subcellular tissues of the lid felt soft to the touch and this bagginess of the lid could be reduced by pressure. The lacrimal gland could be distinctly palpated and could be rolled under the finger, but there did not seem to be a true hypertrophy of the gland structure. This condition had been stationary for the past year or more according to the statement of the mother. The Wassermann, von Pirquet and urine tests were all negative. Patient complained of some slight headaches at times and eyes tire on use.

Vision O.D. 20/20; accommodation type $I\frac{1}{1}$ 7-38 cm.
Vision O.S. 20/20; accommodation type $I\frac{1}{1}$ 7-42 cm.
Refraction under homatropin O.D. + 0.75sp. + 0.25cyl axis 90° = 20/15.
Refraction under homatropin O.S. + 0.75sp. + 0.25cyl axis 90° = 20/15.

Treatment.—The non-operative treatment of the case consisted in the local use of eye-washes and the internal use of Blaud's pill. On March 20, 1913, an operation was done on the right lid. A curved incision was made on the upper lid about 1½ inches long and about ½ inch above the palpebral margin of the lid. The upper border of the incision followed as near as possible the curve of the tarsal cartilage. After careful dissection of the superficial fascia of the lid a yellowish thickened tissue appeared which is never seen in the normal lid. This tissue was about 3 or 4 mm. thick and seemed to extend from directly beneath the skin to the tarsal plate. On continuing the dissection of this tissue a mass of tissue resembling fat pushed itself into the line of the incision and a free opening permitted it to prolapse into the wound. This seemed to extend back into the orbit and could be very nicely traced back and up along the superior wall of the orbit. This mass of tissue was carefully removed. After its removal the superior lobe of the lacrimal gland came forward and as it was thought that it would prolapse more after the removal of the mass of fat, the gland was removed with little trouble and almost no hemorrhage. Great care was exercised to prevent hemorrhage during or after the operation on account of the danger of pressure in the orbit and later on the optic nerve itself. With curved scissors a strip of skin was removed from the upper edge of the original incision and after that a portion of this hypertrophied tissue which was situated between the skin and the deeper tissues of the lid. In suturing the wound a series of buried catgut sutures were put in the deep subcutaneous tissues to prevent, if possible, any tendency to the formation of fat hernia. The skin and the tissues underneath were next brought together with silk sutures to avoid any keloid formation on the scar. A layer of white petrolatum was spread over the incision and stitches and compress bandages were then applied.

The treatment of blepharochalasis, as far as local means are concerned, has been, up to the present time, quite unsuccessful. The local injections of alcohol, iodin and the solution of zinc chlorid has not produced any permanent results. In my first case the electric needle was used with the hope of setting up a cellulitis of the subcutaneous tissues of the lid, with secondary contraction and shrinkage of all of the surrounding tissues, but this did not follow a most persistent use of the needle. The Roentgen ray and the high-frequency current or the violet rays have not been of any service. The use of any general medication has not been followed by any improvement of any of the symptoms. The fact that none of the cases presents any general dyscrasias would seem to be a partial explanation why we do not get any results from this form of treatment.

The only method of dealing with this condition is surgical. Many of the cases have been operated on with a varying degree of success. Simple excision of the skin of the lid with removal of a portion of the skin has not been found to be sufficient to correct the defect. Some of the operations have been followed by what looked like a keloid formation of the scar. Some of the operators have had hernias of the fat and subcutaneous tissues of the lid through the edges of the wound. Rohmer thinks that a special operation is indicated as the esthetic question is the thing that the patients have uppermost in their minds. One must not fear to excise too much and his experience has shown that all of the affected skin area must be extensively resected and even then the result will not be perfect. He has had ugly lumps form which swell at times. If a fatty lump should present during the operation, as much as possible should be removed in order to prevent these hernias of fat as already spoken of. One must exercise great care in the closing of the wound, using catgut sutures and a pressure bandage after placing over the edges of the wound fenestrated silk tissue or petrolatum to avoid any possibility of the dressing adhering to the wound. One of Rohmer's cases he has been able to observe five years after the operation and there is no tendency to recurrence because he removed with his incision all of the affected skin tissue. His other two cases have not been of more than six months' duration at the time of report and therefore no conclusion was justified

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V. 6
1913

ABSTRACT OF DISCUSSION

DR. EDGAR S. THOMSON, New York: I can only speak from the experience of these two cases, both of which I saw with Dr. Weidler. The thing that impressed me most of all was the fact that they remained absolutely stationary no matter what was done. It seems that it is perfectly proper to classify them as dystrophias and I think there is no question that the angioneurotic edemas have no resemblance either in onset or duration. The seton was used after a rather interesting case of facial erysipelas was reported in the *British Medical Journal*. The author describes a thickening of the lids following the erysipelas and mentions the use of the seton through the upper part of the lid and down on the side of the face to establish lymphatic drainage. As Dr. Weidler said, it was unsuccessful in our cases. In view of the operative and microscopic findings, as well as the clinical history, I think operation is the only treatment. I am absolutely in the dark in regard to the nature of this fatty mass. It did not impress me as of the character of a new growth. In fact I think it is well known that it is dangerous to talk about lipoma in the tissues of the orbit unless there is a well-marked new formation. The mass had the appearance of cellular adipose tissue more than anything else. It is possible that, if taken during the edematous stage, the seton might have accomplished more, but later on it certainly seems that operation is the only course. There was marked disturbance, ptosis, swelling, etc., although Dr. Weidler dissected the mass with great care, with a minimum amount of traumatism. It shrank up in a few days and the patient now has good motion of the lid and the case looks very well.

DR. ARTHUR J. BEDELL, Albany, N. Y.: In a girl of 16, American, for at least five years the eyelids have been thickened although her physician believes they have lessened in the past year. Examination of the blood, urine, etc., showed no other organic lesions. Both eyelids present a boggy swelling and the appearance of ptosis. The lacrimal glands are not enlarged. The patient has refused operation and so it is impossible to report on the microscopic findings.

DR. WALTER BAER WEIDLER, New York: I hope that there will be a more careful classifying and reporting of these cases in the future and I believe that there are many more of these cases than a search of the literature reveals. Dr. Lewis told me that he had seen a case which he believed was one of blepharochalasis. Bach, who performed the operation described in the paper, on removing the mass of fat thought it was a true lipoma, but I think that it was only orbital fat. The patient, as Dr. Thomson has stated, had a marked degree of ptosis following the operation and the lid was closed for three or four days afterward; but this condition slowly cleared up and the lids regained the normal position and movements. I would like to warn any one who does this form of operation, to be very careful in inserting the sutures. The first row should begin deep in the orbital tissues and these tissues should be brought well together and the skin edges united with great care. Several of the cases that have had an operation for this condition have been followed by fat-hernias through the edges of the wound and others have seen keloid formations along the line of the scar. In my patient the excision was done first on the right side and about a month later on the left and the results on both sides have been most gratifying and the deformity is entirely removed. I believe that it would be much easier to operate on these cases of blepharochalasis after the swelling has disappeared and we only have to deal with the bagging skin of the lids.

EQUIVALENT VALUES IN SPECTACLE LENSES

WILLIAM E. SHAHAN, A.M., M.D.
ST. LOUIS

By lens-grinders and spectacle salesmen, the powers of spectacle lenses are computed by simple additions and subtractions; a lens which has a + 5 D. surface on one side and + 5 D. surface on the other, is called a + 10 D. lens. Also a lens which has a + 20 D. surface on one side and a — 10 D. surface on the other, is called a + 10 D. lens. But it is found in practice that if this double convex +10 D. lens accurately measures a defect in an eye, and a meniscus lens of this form is ordered with the — 10 D. surface next the eye, an overcorrection of more than 2 D. will exist. In other words this meniscus lens with a conventional value of + 10 D. has a value equivalent to that of a double convex lens of more than + 12 D. Moreover the ordinary neutralization test fails in such a case as this, for if a — 10 sph. lens is held in contact with the convex surface of this meniscus lens, it will very nearly neutralize it. Discrepancies similar to this prevail in all meniscus and toric lenses, and because of the absence of some practical way of easily making compensatory computations the advantages accruing from the use of such lenses have been largely lost, except in the weaker forms.

Most of the principles of changes in the powers and properties of lenses with changes in their forms have long been known to expert mathematicians and physicists, and there are many text-books on geometrical optics from which these principles can be extracted by expenditure of sufficient time and labor. Even in the most modern text-books on ophthalmology, however, there can be found scarcely an echo of the progress made in geometrical optics since the work of Gauss (1841) or even that of Wollaston (1803). It is with a view to bringing to practical usefulness some of these principles that the present work is attempted. Because of the widespread looseness in defining focal lengths in text-books on ophthalmology, an elementary description of the usual method of geometrical optics will be here attempted.

Parallel rays of light near the axis, striking the left surface of a positive lens, as in Figure 1, come approximately to a point on the other side. This point is universally recognized as the second principal focus of the lens. Likewise similar rays striking the right surface come to a point on the left side of the lens in the first principal focus. Either of these points is universally agreed on as one of the points from which to measure focal lengths. It is with regard to the selection of the other point that so much confusion exists. This other point was definitely and accurately fixed by Gauss through observation of the behavior of the image as an object approaches a lens. If an object is very far to the left of a positive lens there will be a small inverted image of it very near the second principal focus. As the object approaches the lens the image moves away and becomes larger. When the object reaches a certain point twice the focal length of the lens on the left side, the image will occupy a corresponding point on the opposite side where it will be exactly the same size as the object, but still inverted.[1] As the object continues to approach the lens the image continues to recede from it and grows larger. When the object is in the first principal focus, the image is very far away and very large. When the object passes the first principal focus, so as to get between it and the lens, the image changes to the left side of the lens, is very large, very far away, virtual and upright. As the object continues to approach the lens the image rapidly approaches the object, becoming smaller. It finally catches up with the object and passes it, getting smaller, until a point is reached where the object and image are again of the same size, the image remaining upright and virtual. The planes occupied by the object and image in this position, where both are upright and of the same size, are known as the first and second principal planes of Gauss, and the points where they cut the axis as the first and second principal points of Gauss. These are most commonly referred to simply as the first and second principal planes and points. These planes and points can always be accurately located both by experiment and mathematical computation. In lenses in air the first principal point is always the same distance from the first principal focus as the second principal point is from the second principal focus. One of these principal points is now universally agreed on, except in text-books on ophthalmology, as the other point from which to measure focal lengths. The focal length of a lens in air is then defined in this paper as the distance between either principal focus and the corresponding principal point. There are two other points which in a lens in air coincide with the principal points. These are the nodal points, and in text-books on ophthalmology focal lengths are frequently stated to be distances between principal points and nodal points. This should be especially guarded against, as it may lead students into believing that the focal lengths of the eye itself are measured from nodal points to focal points.

With this understanding of principal points, focal points, and focal lengths, we are now in a position to

1. The principal planes of Töpler.

study changes in these points and lengths brought about by changes in the bounding surfaces of a lens.

first, (*A*) is a double convex lens with each of its surfaces

In Figure 1 are shown four lenses whose rims are in the same plane, as shown by the dotted line (1). The equal to + 5 D. It will be noted in this lens that the principal planes 1, 2 are within the glass symmetrically placed on each side of the center. The second principal focus of this lens is in the dotted line (2). The second (*B*) is a convexo-plane lens. Here the first principal point is in the curved surface of the lens, while the second principal point is within the glass about two-thirds of the thickness of the lens in front of the plane surface. The second principal focus is in front of the dotted line (2) and therefore nearer the dotted line (1). This + 10 D. lens, then, when its curved surface is turned toward the incident light, has the effect of a lens somewhat stronger than the + 10 D. lens *A*. The third (*C*) is a meniscus lens of which the anterior surface is + 21 D. and the posterior surface is — 11 D. In this lens the first and second principal points are both in front of the anterior surface of the lens. The second principal focus is considerably in front of the dotted line (2). This lens, therefore, when its convex surface is turned toward the incident light, has the effect of a lens of notably greater strength than that of the lens *A*. The fourth (*D*) is a meniscus lens of focal length equivalent to that of the double convex lens *A*.

The effective strength of each of these lenses depends on the distances through which the second principal points are advanced from the plane of the rims of the lenses (dotted line), and on a certain amount of absolute shortening of the focal length as the lens assumes the meniscus form. If, now, some way can be devised for calculating the actual focal length of the lens and the distance through which the second principal point advances as the lens changes its form, it will be easy to find its effective or equivalent value in terms of any other lens.

In this paper lenses are said to have the same equivalent value when parallel rays of light near the axis are brought to a focus at the same distance from the plane of the rim of the lens. As will be seen later, this change in position of the second principal point will have to be computed in two parts: the distance of the second principal point from the apex or pole of the posterior surface of the lens, and the distance of the pole of the second surface from the plane of the rim of the lens.

In order that the properties of a lens may be discussed in a mathematical way it is necessary to have at hand certain essential facts: the refractive index of the glass of which the lens is made, the thickness of the lens, and the radii of curvature of its bounding surfaces. This last involves a knowledge of the radii of curvature of the tools with which these surfaces are ground, and with which they coincide. The glass used by my opticians happens to be what is known as "B & L" glass and the refractive indices for various rays of the Fraunhofer scale were kindly furnished me by the Bausch and Lomb Optical Co. The refractive index of the Fraunhofer ray D. is generally used in geometrical optics as the standard. In this case it is 1.5225. The thickness of all positive lenses, except where the radii are equal, can be computed if the diameter of the lens and its radii of curvature are given. In this paper the greatest diameter (the major axis in elliptical lenses) of all lenses is fixed at 40 mm. which corresponds to the "00 eye" of the opticians.

The radii of curvature of the surfaces of the lenses can be determined from a consideration of the tools by which they are ground. Each tool is adapted for one fixed surface and the number of surfaces which the ophthalmologist can dictate therefore is limited by the number of individual tools which his optician possesses. The radii of curvature of these tools are computed from the ordinary formula for refraction at a spherical surface: $f = \frac{r}{n-1}$ where f is the focal length of the surface measured from the apex of the surface, r the radius of curvature of the surface and n the index of refraction of the glass. Now if D = the power of the surface in diopters, then $D = \frac{1}{f} = \frac{n-1}{r}$ Since n = 1.5225 in this paper, $D = \frac{0.5225}{r}$ in meters, or $D = \frac{52.25}{r}$ in cm., whence $r = \frac{52.25}{D}$ cm. From this last formula by substituting the dioptric value D of a surface of any lens ground with "B & L" tools, the radius of curvature of that surface can be found. For example if the lens has a — 10 D. surface on one side, the radius of curvature for that side will be $r = \frac{52.25}{10} = 5.225$ cm. For the long series of computations made in this work these radii were computed at diopter intervals up to 30 D. and the results tabulated for rapid reference.

Now let t be the thickness of a plano-convex lens, t′ that of a double convex, and t″ that of a meniscus lens. It can be shown by simple geometrical methods that $t = r - \sqrt{(r-y)(r+y)}$, $t' = 2r - 2\sqrt{(r-y)(r+y)}$ and $t'' = r - \sqrt{r^2-y^2} - (r' - \sqrt{r'^2-y^2})$ where r is the radius of curvature of the anterior surface, r′ the radius of curvature of the posterior surface and y the semidiameter of the lens. The last equation is merely the difference between two plano-convex lenses. So that when the thicknesses of the 26 possible whole-diopter planoconvex lenses, with semidiameter y = 2 cm. have been calculated, the thicknesses of the 325 possible whole-diopter meniscus lenses with semidiameter y = 2 cm. can be obtained by simple successive subtractions. This was done and the results tabulated for ready reference. To all these minimum thicknesses 1 mm. is arbitrarily added as an approximation of the thickness the optician will allow at the edge of the lens.

This gives all the data necessary for the computation of focal lengths, equivalent focal lengths, powers and equivalent powers of any given form of lens in terms of any other form. If H′ represents the distance of the second principal point in front of the apex of the posterior surface of the lens, d the distance of the apex of the posterior surface of the lens in front of the plane of the rim of the lens, F the focal length of the lens and E F the equivalent focal length of the lens, then E F = F — (H′ + d). That is, the equivalent focal length is the distance from the plane of the rim of the lens to the second principal focus. Now $F = \frac{-r\,r'}{(n-1)(r-r'-\frac{(n-1)t}{n})}$ $H' = \frac{-\frac{r'}{n}t}{r-r'-\frac{n-1}{n}t}$ and $d = r' - \sqrt{(r'-y)(r'+y)}$ which is the thickness of a planoconvex lens, fitting accurately into the concavity of the meniscus lens.

When these values are substituted in the equation E F = F — (H′ + d) the equivalent value of a meniscus lens in terms of a double convex lens is obtained. Since

* Adapted from Heath's Geometrical Optics, Second Edition, page 69.

V. 61 1913

E F is in centimeters the equivalent value in diopters will be $\frac{100}{E F}$. In double convex lenses the second principal point is slightly to the right of the plane of the rim of the lens. So that a small quantity $\frac{H H'}{2}$, where H H' is the distance between the principal points is neglected in the equation $E F = F - (H' + d)$. Since planoconvex and meniscus lenses are always set nearer the eye than double convex lenses, it is more accurate for this quantity to be neglected. If now a fixed value, say — 11 D is assigned to the posterior surface of a lens, and its anterior surface allowed to vary by diopter intervals from + 11 D to + 25 D (which is the maximum for a lens where y = 2 cm. and N D = 1.5225) two sets of values will be obtained: first, the ordinary conventional values obtained by simply subtracting one surface from the other, and second, the equivalent values obtained from the equation $E F = F - (H' + D)$.

More than two hundred independent solutions of the equation $E F = F (H' + d)$ were required to obtain sufficient equivalent values for use as points in the construction of curves from which all equivalent values might be readily read off. These solutions were made

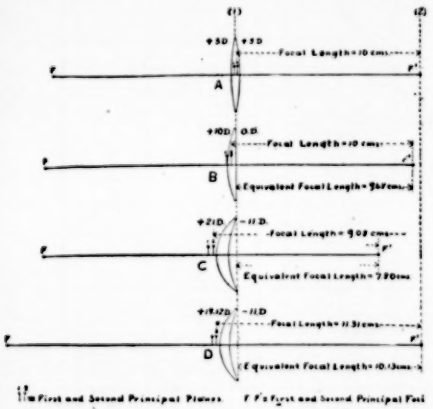

Fig. 1.—First and second principal planes and foci.

with seven-place logarithms taken from Vega's tables, and each point calculated to the thousandth part of a diopter. Each series of points for each meniscus lens of fixed posterior surface was then laid out on coordinate paper, and connected by a smooth continuous line through the agency of a spline and a number of weights. The conventional values of all meniscus lenses are plotted along the left vertical margin of each chart and the corresponding computed equivalent values for double convex trial case lenses are plotted along the lower horizontal margin of each chart.

PRACTICAL RULES FOR USING THE CHARTS

Figure 2 is a general chart giving equivalent values for all meniscus lenses with posterior surfaces ranging from zero to —20 D. This is especially useful for high powers. Figure 3 is a special chart for convenience in reading off equivalent values for the common commercial meniscus whose posterior surface is —6 D and for ordinary toric lenses whose common base is 6 D. The divisions in both these charts are diopters, halves, quarters and eighths of diopters. Figure 4 is a general chart giving equivalent values to 6½ diopters only and the divisions are diopters, halves, quarters, eighths and thirty-secondths of diopters. This is designed for determining equivalent values for weak lenses in order to decide whether or not the differences are small enough to neglect with safety. Each type of lens will now be taken up in detail.

Double Convex Lenses.—If a double convex trial case lens is used, and it is desired to find an equivalent meniscus, first decide what the posterior surface is to be, then seek out along the lower horizontal margin of any of the charts the number representing the dioptric value of the lens used in the trial measurements, next follow the vertical line leading from this point directly upward to the curve marked P. S.= the chosen posterior surface, then follow the nearest horizontal line leading from this point to the left margin and add to the number found there the numerical value of the chosen posterior surface; the sum of these two numbers is the dioptric value of the front surface of the equivalent meniscus. For example, suppose that the patient has an aphakial eye and his defect is measured by a + 12 double convex trial case lens and it is desired to prescribe a meniscus lens whose posterior surface is — 11 D. Using Figure 2, the number 12 is found on the horizontal margin under the heading "Equivalent Values of Double Convex Trial Case Lenses." The line leading from there is followed up to the curve marked P. S.= — 11. From this point the nearest horizontal line is followed to the left vertical margin marked "Conventional Values of Meniscus Lenses." The number found there is 9.50. This added to 11 gives the desired anterior surface. The meniscus with — 11 D posterior surface equivalent to a + 12 D double convex lens has therefore an anterior surface of + 20.50, so that the prescription for the optician would be:

R	Back surface	—11 sph.
	Front surface	+20.50 sph.

Other examples are:
For double convex + 12 sph.:

R	Back surface	—14.50 sph.
	Front surface	+23 sph.

For double convex + 15 sph.:

R	Back surface	—12 sph.
	Front surface	+23 sph.

For double convex + 20 sph.:

R	Back surface	—10 sph.
	Front surface	+24 sph.

In all cases "B. & L." glass on "B. & L." tools "00 eye" with greatest diameter = 40 mm.

Planoconvex Lenses.—Although computations for points used in constructing the charts were made for equivalent values in terms of double convex lenses, equivalent values in terms of planoconvex lenses can be read off with equal facility. This arises from the fact that a planoconvex lens is nothing more or less than a meniscus lens whose posterior surface is zero. The first curve in all the charts is, therefore, a planoconvex curve. Moreover, the conventional value of a planoconvex lens is also its actual value. If, then, an aphakial eye requires a + 12 D planoconvex lens to bring its vision to a maximum, and it is desired to prescribe a meniscus lens with P. S.= — 11, we find on the left vertical margin of Figure 2 under "Conventional Values of Men-

iscus Lenses" (remembering that these are the actual values of planoconvex lenses) the number 12, and follow the horizontal line leading from this to the curve marked P. S.= 0; from this point we drop directly downward to the curve marked P. S.= — 11; from this point we follow the nearest horizontal line back again to the left margin and add the number found there to the number 11 for our front surface. The nearest number found there is 9.87. This added to 11 gives + 20.87 for the front surface. The nearest tool the optician has to this number is + 21, so that our prescription would be:

℞ Back surface..... —11 sph.
Front surface..... +21 sph.

wholly on the ability with which the practical lens grinder can fashion such surfaces. At one time there was a variety of basic curves used for toric surfaces, but opticians have now quite generally settled down to a 6 D curve on which cylindric curves are superimposed. Moreover, it has been found that fewer mechanical difficulties beset the grinding of positive than negative toric surfaces. In consequence of this, unfinished lenses which are sold in wholesale quantities to retail opticians have on their front surfaces + 6 D sph. with any desired cylindric increase in one meridian. If it is desired to order, say + 7 sph. + 3 cyl., and a toric lens is designated without further specification, the finished lens will have on its front surface +6 sph. + 3 cyl. and on its back surface + 1 sph. This sacrifices the chief

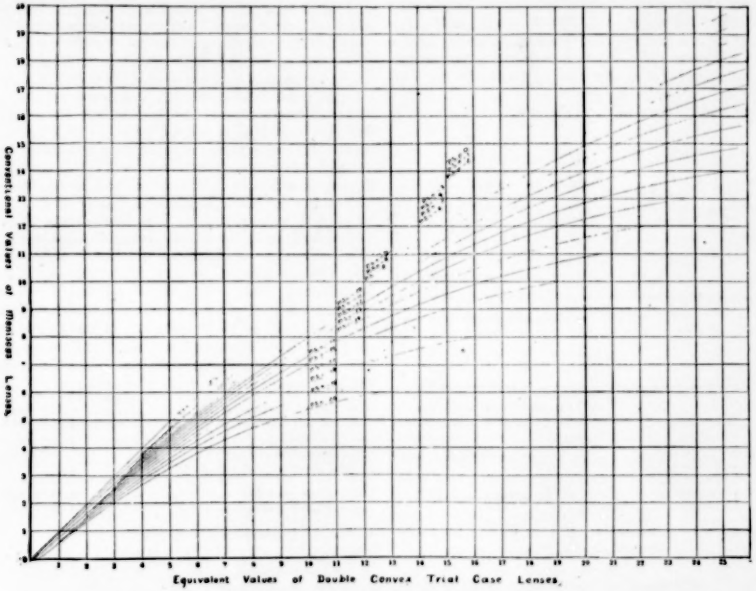

Fig. 2.—Equivalent values in spectacle case lenses.

It will be seen that this method of finding equivalent values of planoconvex lenses is the same as finding the values of the planoconvex lenses in terms of double convex lenses and then using these values as if double convex lenses had been used in making the trial tests. Other examples are:

For planoconvex + 15 sph.

℞ Back surface..... — 6 sph.
Front surface..... +19.50 sph.

For planoconvex + 6.50 sph.

℞ Back surface..... —18 sph.
Front surface..... +22.50 sph.

Toric Lenses.—Lenses having toric surfaces have been designed in order to extend to patients afflicted with astigmatism the benefits of meniscus lenses, and the degree to which such benefits can be extended will depend

advantage, the meniscus effect, of toric lenses of this strength. If, therefore, the full value of the increased cost of toric lenses is to be secured for a patient it becomes necessary to dictate both surfaces.

In taking from the charts equivalent values for toric lenses, discrepancies arising from the interval between the two trial lenses used in making the refractive measurements are, as usual, neglected and the trial combination considered as if composed of one lens with its meridians of unequal power. The equivalent value of the two principal meridians can then be taken as if each were a separate lens. Suppose, for example, that the patient's refractive defects are corrected by + 10 sph. (double convex) + 4 cyl. ax. vert. Knowing that your optician's toric tools are on a 6 D base, you can at once write down:

V. 61
1913

Back surface — 6 sph. — 4 cyl. ax. hoz. That is, one meridian which is — 6 D and another which is — 10 D. Now, one of the meridians of your trial combination is + 10 D and the other is + 14 D. The weakest negative meridian is always behind the strongest positive meridian. Accordingly the two meridians can be considered as two lenses for which equivalent values are desired. One of these is the equivalent of a + 10 D double convex with P. S.= — 10 D and the other a + 14 D double convex with P. S.= — 6 D. From Figure 2 we find for the equivalent of + 10 double convex lens with P. S.= — 10, front surface = + 18.37 and for the equivalent of a + 14 double convex lens with P. S. = — 6, front surface = + 18. The front surface of the finished lens is, therefore, between + 18.37 sph. and + 18. sph., compromising on + 18.25. The prescription will be:

℞ Back surface..... —6 sph. —4 cyl. ax. hoz.
Front surface..... +18.25 sph.

If the trial lenses were planoconvex lenses the prescription would be:

℞ Back surface..... —6 sph. —4 cyl. ax. hoz.
Front surface..... +18.50 sph.

The front surfaces for the two meridians are so nearly identical that in practice it will be necessary to find the front surface for only one meridian and use that for the front surface of the finished toric lens, using Figure 3, therefore, equivalent toric lenses in terms of either planoconvex or double convex trial lenses can be obtained by the following simple rule: Write down at once for the back surface — 6 sph. with a minus cylinder at right angles to the plus trial cylinder (if a plus cylinder was used). Then find the equivalent front surface of the lens of power equal to the strongest meridian of the combination used with P. S.= — 6 and write this down for the front surface of the toric lens. For example: For + 8 sph. (double convex) + 2 cyl. ax. 45°.

℞ Back surface..... —6 sph. —2 cyl. ax. 135°
Front surface..... +15 sph.

For + 8 sph. (planoconvex) +2 cyl. ax. 45°.

℞ Back surface..... —6 sph. —2 cyl. ax. 135°
Front surface..... +15.25 sph.

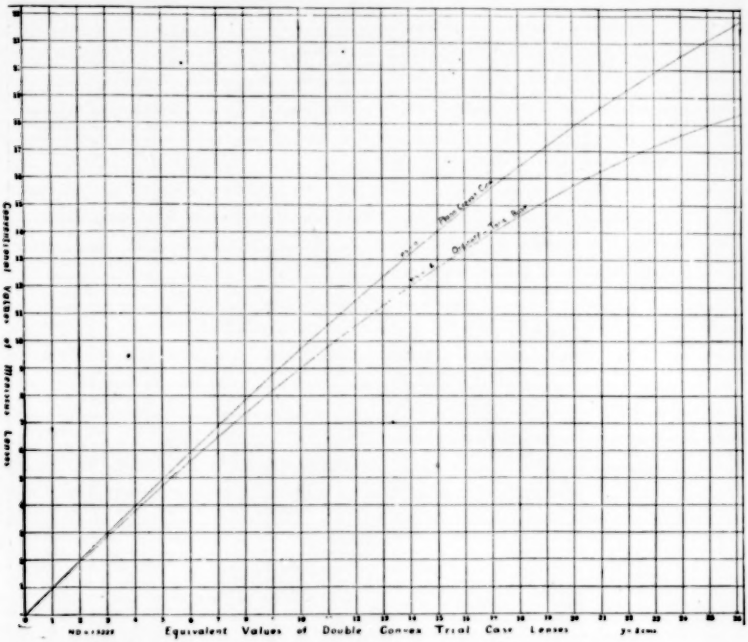

Fig. 3.—Equivalent values of double convex trial case lenses (for common toric lenses).

In both these cases the strongest meridian is + 10 D. In the first case the number 10 is found at the bottom of Figure 3; the vertical line followed from there to the curve P. S.= — 6 and the horizontal line followed from that point to the left margin and the number 6 added to the number 9 found there gives the front surface. In the second case the number 10 is found on the left margin of Figure 3, and the horizontal line followed from there to the curve P. S.= 0. Then the nearest vertical line is followed directly downward to the curve P. S.= — 6. From this point the nearest horizontal line is followed back to the left margin, and the number 6 added to the number 9.25 found there gives the front surface. This is exactly the procedure

already described for simple lenses. With a little practice this can be done with ease and speed.

The variety of meniscus and toric lenses that can be ordered is practically unlimited. Fractional values for posterior surfaces are obtained by interpolation. The "back surface —14.50" shown, for instance, was obtained by choosing a point on the vertical line half way between the curves marked P. S.——14 and P. S.——15. Variations in the diameter and thickness of the lenses will cause variations in their values; but these are generally small enough to be adjusted by having the spectacle lens set a little nearer or a little farther away from the eye, as determined by actual tests of visual acuity with the finished lens.

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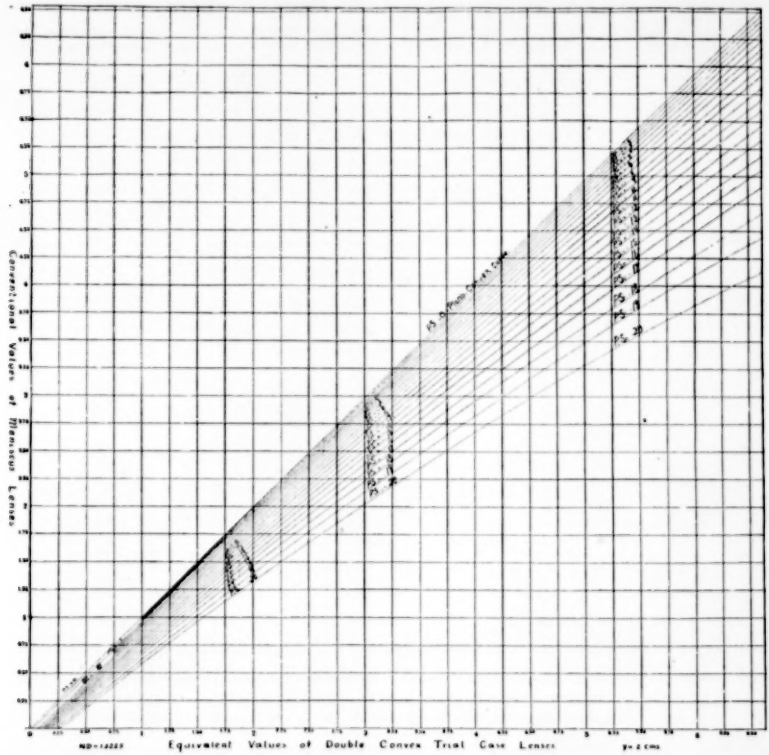

Fig. 4.—Equivalent values of double convex trial case lenses (for weak lenses).

ABSTRACT OF DISCUSSION

DR. WILLIAM R. MURRAY, Minneapolis: In estimating the equivalent value of a meniscus lens, as compared to a biconvex lens of a given diopter strength, we must take into consideration the relative position of the plane of the rim of the lenses in reference to the apex of the cornea. In practice, if the rim of a plus meniscus lens of 10.00 D, having a posterior surface of —10.00 D, be placed in the same plane as the rim of a plus 10.00 D, biconvex lens, there will be an overcorrection amounting to about 2 D. Also if a —10.00 D sphere be placed against the anterior convex surface of this meniscus it will neutralize it, as the posterior principal point of the biconcave sphere very nearly coincides with the second principal point of the meniscus. Theoretically the two lenses are of equivalent strength, as the focal length is the same in both lenses. It is then a question of the relative position of the two lenses in reference to the eye, as to whether they are equivalent in practice. As we cannot bring the meniscus lens near enough to the surface of the cornea to make the second principal point coincide with the second principal point of the biconvex we find that the two lenses are not equivalent and some method of computation is necessary whereby we can determine the equivalent value of a meniscus lens in the position which it will occupy in reference to the eye. The overcorrection which would result if a plus 10.00 meniscus were substituted for a plus 10.00 biconvex lens, with the rim of the lenses in the same plane, would be partially eliminated, if the plane of the rim of the meniscus is brought near enough to the eye to allow the apex of the posterior surface of the meniscus to occupy about the same plane as the apex of the posterior surface of the biconvex. The two practical points to be considered in determining the

equivalent value of a meniscus as compared with a double convex lens are the distance between the apex of the posterior surface and the plane of the rim of the lens, and the distance between the apex of the posterior surface and the second principal point. The first point can be largely eliminated, owing to our being able to bring the plane of the rim of a meniscus of high diopter strength considerably nearer the eye than the plane of the rim of a biconvex of the same strength. The second point becomes one of practical importance as the focal length of a lens is measured by the distance between the second principal point and the second principal focus and in a meniscus lens of high diopter strength, as compared with a biconvex lens of the same diopter strength. In a plus meniscus lens of 10.00 D, with a posterior surface of minus 11.00, the second principal point lies near the anterior surface of the lens and outside of the lens; in a plus biconvex lens of 10.00 D, the second principal point is within the lens and posterior to the plane of the rim of the lens. When the meniscus is brought into its proper position, in reference to the eye the second principal point will still be anterior to the second principal point of the biconvex lens, in its proper position before the eye, and this difference will approximately equal the thickness of the biconvex lens. It becomes necessary to make a certain allowance in estimating the equivalent value of the meniscus lens and any method of computation by which this equivalent value can be furnished for ready and quick reference is of practical value.

There is no doubt but that, in many cases, the meniscus lens offers many advantages over the biconvex or plano convex lens and in order to obtain the maximum advantages of a meniscus in certain cases, as in cataract lenses, we should be able to prescribe the degree of concavity of the posterior surface. In this country the opticians are furnished with plus menisci lenses having a posterior surface of minus 1.25 and minus 6.00. Plus menisci lenses having a posterior surface of minus 9.00 may be obtained but at a considerable increase in cost, while if we wish to prescribe a lens having a posterior surface of minus 10.00 or minus 11.00 it requires special grinding at much greater cost.

This is somewhat of a disadvantage, at present, to our prescribing such lenses and means should be devised whereby menisci lenses of the most suitable degree of concavity can be obtained at a reasonable cost.

DR. F. PARK LEWIS, Buffalo: It is quite evident that the difference between the bifocal lens and the meniscus lens must, in the higher degrees, affect the values. We have all known that the optical center of the lens is of importance in relation to the eye, but we have usually made the correction by approaching the stronger lenses closer to the eye or withdrawing it somewhat further from it. Dr. Shahan's method, even when his chart is closely followed, would necessitate an approximation of values. The question arises: Why should we not have our test lenses made as are our ultimate prescriptions, that is, in menisci. We have all been annoyed by the discrepancies between the optician's lenses and the prescription which we have made, and the seeming discrepancies would be explained by the fact that our tests are made with bifocal lenses while our final prescription is in toric form. A second point is this. If, as Dr. Shahan has pointed out, a discrepancy, in the stronger lenses, as great as 2 diopters may exist between the test lens and that which the patient receives, and we have only Dr. Shahan's chart to enable us to determine how great this discrepancy is, must not our final result in each instance be merely an approximation? The opticians have certain forms on which their lenses are ground. They cannot give us 19¼ or 19½ on one side, and unless in special lenses we should have special forms made which would be so expensive as to be impracticable, we must still vary our prescription from our findings. The subject is one of great importance, and the refractionist and the optician should certainly work together in order that it may be possible for the former to secure an exact reproduction of his office test in the final lens.

DR. WALTER BAER WEIDLER, New York: I should like to ask Dr. Shahan whether this applies only to the higher degrees of lenses, or whether the condition which he found with his patients was also present in the lower degrees of refractive errors. In my own personal experience in attempting to wear toric lenses, to determine the additional values as claimed by the optical men, I found that I was very uncomfortable and absolutely unable to wear them. They had the very distinct advantage of giving a greater field of vision but on account of my own discomfort I have been averse to prescribing toric lenses.

DR. GEORGE H. PRICE, Nashville: There is a popular idea that toric lenses are by far the best, and the judgment is that this is true theoretically. Practically they fall far short of what we desire, and patients are not infrequently disappointed, because of the facts brought out in this paper. They are disappointed because the ratio of increase from the smaller or lower power to the higher power is rapid and this increase in the refractive capacity of the lens induces marked symptoms. I once had a irritation in my eyes, and going to a drug-store, bought a pair of colored glasses. They were toric, pressed lenses. I noticed when I walked with them that everything seemed to be out of place—things were distorted and they induced nausea. The nausea would increase until I would have to take the glasses off. After a while I could wear them. My experience in prescribing torics, for patients who have worn biconvex or plain lenses or simple myopic lenses, has shown that when they put on toric lenses they return with a whole train of nervous symptoms not altogether easily explained. I have one patient now who has been accustomed to wearing glasses for some years. She wanted toric lenses because of the increase of the field of vision; but it has taken her now nearly three months to adjust herself to the toric lenses. They are very low in value, the strongest being 1 diopter and the other ½ diopter. These symptoms are very annoying. It is a great advantage to have a chart from which one can estimate the lenses. The low powers are not so distressing as the high powers. I read with toric lenses at night and if I don't feel well it makes me sick, though I do not have a high defect of vision. It is this constant draft on the nervous mechanism to adjust the eye to the lens itself which gives the distressing reflex disturbances, and with this chart we can properly correct them.

DR. JOSEPH S. LICHTENBERG, Kansas City, Mo.: Professor Gullstrand of the University of Upsala has worked up the formula for aspherical meniscus lenses to be used especially in cataract cases. They are made at present only by the well-known Zeiss firm in Jena, Germany. The data required by the lens grinders are: (1) the form of lens, biconvex or planoconvex and its strength in diopters; (2) whether in testing, the spherical or cylindrical lens was nearer the eye; (3) the strength in diopters and the axis of the cylindrical lens; (4) the distance from the summit of the cornea to the posterior surface of the lens nearest the eye and (5) the distance between the anterior surface of the lens nearest the eye and the posterior surface of the lens farthest removed from the eye. I find they are very satisfactory to my patients, as they have at least 60 degrees of field, which is a great deal better than any other lens we can obtain.

DR. J. A. DONOVAN, Butte, Mont.: The Bausch and Lomb people furnish lenses with one refractive index and other makers another and we get confused in the index of the lens we are using. We are now using two normal standards in the indices of these lenses. Will it be necessary to have a table for each?

DR. WALTER B. LANCASTER, Boston: A lens of the correct strength is not sufficient; it must be mounted the proper distance from the eye. After calculating exactly the lens we want to use and having prescribed it, the practical point is that when the patient comes in with that lens we must try it out in our offices and see whether slight changes in the distance from the eye are needed or whether putting ¼ or ½ or even ¼ of a diopter in front will improve things. If it does we must either change the prescription or slightly change the distance of the lens from the eye. In many cases we must slightly tilt the frame to get the best correction for astigmatism. The frame furnished by the optician may vary a few degrees which can be corrected by slightly bending the frame.

DR. WILLIAM E. SHAHAN, St. Louis: I was induced to work this chart out on finding that conventional meniscus and

toric lenses give overcorrection, so that the power of the lens must be reduced. The ordinary optician does not understand how to do this. In all branches of physical science it is necessary to have some definite point from which to make exact measurements. Because of this, it is best to keep the rim of the trial frame as nearly as possible in the position which the spectacle or eyeglass rim is expected to occupy. Gullstrand lenses have some advantages over ordinary meniscus or toric lenses but are more expensive, and more time is consumed in obtaining them. Meniscus trial case lenses probably would not be successful because of the varying positions of the principal points and the difficulty of combining them with cylinders. Planoconvex trial case lenses are probably the best possible form. The charts are based on glass with refractive index 1.5225. Other charts will have to be constructed for glass of other refractive index. Toric lenses unquestionably require greater care and skill both in fitting and in grinding than ordinary types. If, for instance, the center of the lens is not accurately set in front of the center of the pupil, some astigmatism of axial pencils will be obtained. If the lens for one eye is accurately centered and the other is not, a condition of anisometropia will be introduced. Our duty to our patient is never completed until we have seen him with the finished lens, taken his vision both through the center and periphery of the lens near each end of the major axis and searched for any possible residual defect. Other questions asked are covered in the paper.

METASTATIC OPHTHALMIA

REPORT OF THREE CASES, ONE OF WHICH RESULTED IN RECOVERY OF VISION

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Professor of Ophthalmology, Rush Medical College in Affiliation with the University of Chicago
CHICAGO

"Metastatic ophthalmia" is the name given to a serious inflammation of the structures of the eyeball that results from an endogenous infection of the retina or uveal tract. "Metastatic suppurative chorioiditis" is another term used for the condition, but as the septic process so frequently begins in the retina, and so frequently involves all the structures of the eyeball, the name "metastatic ophthalmia" would seem preferable. "Septic endophthalmitis" is a good name for the same condition, but even this does not indicate accurately the grave features of general inflammation in the severe cases, which resemble panophthalmitis from traumatic or ectogenous infection of the globe.

I desire to present a report of three cases of this disease, in one of which the result was so unusual as to make it of especial interest.

CASE 1.—Oct. 3, 1910, I was called to see Mrs. H., aged 64, in consultation with Dr. William Cuthbertson and Dr. Capps. A few days before she had noticed a rather sudden failure of vision of the left eye, which had been preceded by a blurring or fogginess of the sight. She had had no previous evidence of glaucoma. The conjunctiva also showed considerable redness and some chemosis and the eyelids of that side became swollen, but not accompanied by pain.

She had been under treatment for cystitis for some time, and pus and albumin were found in the urine. Temperature was 102; blood-pressure 220 mm. There was a painful swelling of the right parotid gland which had developed a day or two before the time I first saw her. There was also a painful swelling of the left leg, which seemed to be due to thrombosis. The condition was that of general sepsis; there were no physical signs of endocarditis.

At the time of my first visit, the vision of the right eye was fairly good, but that of the left was zero. In the left eye there was marked chemosis with considerable dusky redness of the conjunctiva. The lids were also considerably swollen and there was some proptosis. The cornea and anterior chamber were clear, the pupil about 3 mm. in diameter and immobile, the lens clear and a greyish reflex was obtained from the vitreous with the ophthalmoscope. Tension was increased.

In the right eye there was no redness or chemosis of the conjunctiva; the cornea was clear, but the anterior chamber was rather deep. Pupil was clear and about 3 mm. in diameter. The vitreous was faintly hazy, but an ophthalmoscopic examination could be made and the optic disk seemed slightly reddened, the arteries small and the veins engorged. No hemorrhages and no plaques of exudate or degeneration were seen.

The next day, October 4, the condition of the eyes was worse. The sight of the right had failed rapidly, some chemosis had appeared, and the fundus was invisible because of the cloudy vitreous. Pupils had not dilated with a mydriatic. There was slight hypopyon in the left eye.

General condition was somewhat improved. In two days the right eye was as bad as the left and was totally blind. There was no great pain in either eye, the chemosis was not intense nor did the inflammatory process go on to the extent of making it necessary or wise to open the eyeball.

Dr. Capps found *Bacillus coli communis* in cultures from the urine, and from the blood *Staphylococcus aureus.*

The inflammatory process in the eyes was gradually subsiding, both corneae remaining clear, when the patient died from terminal pneumonia, presumably of embolic origin. Autopsy was not obtained. Death occurred twelve days after the infection of the eyes.

In this case there was a septic metastatic endophthalmitis probably caused by emboli of staphylococcus such as were found in the blood. Probably the same organisms caused the thrombosis in the leg. Although at one time the general condition of the patient seemed to be improving, the development of the disease so rapidly in the second eye indicated the profound nature of the sepsis and the grave prognosis.

CASE 2.—J. A., aged 25, a well-developed young woman, early in January, 1912, had a severe tonsillitis, following which there was a suppurative otitis media of the right ear. Some weeks afterward she developed a pneumonia from which she recovered slowly; elevated temperature continued, with occasional chills, headache and general malaise. There was also severe pain in the head and it was thought she was developing a brain abscess, from the infection of the ear. The left wrist and the left knee became swollen and painful.

Feb. 25, 1912, Dr. Thomas Roberts referred her to my service at the Presbyterian Hospital and the following condition was noted: There was marked edematous swelling of the lids of the left eye. The conjunctiva was very red and there was considerable chemosis. The cornea was clear; the pupil was partially dilated, and lens was clear, but the vitreous was so cloudy that the details of the fundus were not visible. Tension was slightly minus. There was considerable pain in and around the left eye and some swelling of that side of the face. I was told these symptoms of the eye had developed three days previously beginning with a cloudiness of vision which had increased so that at the time of my examination she could only perceive light with that eye. The right eye was in every way normal.

Temperature was slightly elevated, being 100 and pulse was 90. There was still some pain and slight swelling of the left wrist and the left knee. The right ear was still discharging through a large perforation in the posterior-inferior part of the membrane. Dr. E. C. Rosenow made smears and cultures from the tonsils, the right ear and the conjunctiva of the left eye, and these showed streptococci of a highly virulent, moderately hemolytic, encapsulated type. They resembled organisms that he had recovered in tonsillitis and erysipelas cases of an epidemic character that had developed where a certain milk had been used. No organisms were found in the blood. In the conjunctiva were also large numbers of pseudodiphtheria bacilli.

The active symptoms gradually subsided, but in a few days there was no longer perception of light, and the eyeball began

V. 6
1913

to shrink. A yellowish exudate could be seen filling the vitreous chamber. The pupil was fixed but the lens remained clear.

The treatment included irrigation of the conjunctival sac with boric solution, atropin and hot applications. Hexamethylenamin (urotropin) was also administered, 5 grains three times a day. When recently seen the eye was considerably shrunken but quiet.

This case illustrates one of the milder forms of the disease, but was probably caused by one of the virulent forms of streptococcus. Although the inflammation of the eye was not so intense as that of the preceding case, the general septic condition was very severe. The infection may have gained entrance through the tonsil or through the ear.

CASE 3.—Marion B., aged 18, a strong, healthy girl had a chill on Feb. 11, 1913, with headache and vomiting. The physician who first saw her found the tonsils inflamed, for which he gave treatment. A slight membrane appeared on the tonsils and a culture from this made by the Chicago Laboratory showed diphtheria.

Feb. 15, 1913, the physician injected 4,000 units of diphtheria antitoxin after which the throat began to clear, and two days later a second dose of 4,000 units was administered. At this time it was noticed that both eyes began to get red and the following day, February 17, she had dimness of vision and considerable pain in both eyes.

February 18, vision was reduced to perception of light and the eyes were congested and painful. At the request of Dr. Febb, who had been called in attendance, I was consulted at this time and the case was seen by my associate Dr. Darling, who prescribed atropin, dionin and hot applications. A smear from the conjunctiva showed numerous staphylococci.

February 20, the patient was brought to the Presbyterian Hospital where I first saw her. There was marked ciliary congestion in both eyes and some chemosis. The pupils were beginning to dilate from the atropin that had been used for two days, and there was considerable pain in the eyes and in the temporal regions. There was a dense exudate in the pupil covering the central portion of the anterior surface of the lens, and a slight red reflex could be obtained around this, but the vitreous was cloudy and the details of the fundus could not be seen. She could perceive hand movements with each eye. Patient had temperature of 100; pulse 90. A subconjunctival injection of 1 c.c. of physiologic salt solution in each eye, hot applications, dionin and sodium salicylate (15 grains every three hours) were ordered. The next day the condition of the left eye was somewhat improved, the pupillary exudate was less and the pupil was dilated, but the vitreous was very cloudy.

The right eye had not changed very noticeably except that the pupil was slightly wider. Vision of the left eye was better, for she could distinguish forms. Cultures made from the tonsils by Dr. E. C. Rosenow showed *Streptococcus viridans*, and from the conjunctiva staphylococci were obtained. Examination of the blood for organisms gave a negative result.

The subconjunctival injections of salt were repeated and the throat was ordered swabbed with hot 33 per cent. alcohol several times daily followed by 25 per cent. argyrol. Irrigation of conjunctiva with 1:5,000 mercuric chlorid every four hours followed by application of 1:3,000 mercuric chlorid ointment.

Feb. 22, 1913, considerable reactions from the injections the day before and some chemosis were present, but the condition of the eyes was greatly improved, for the pupillary exudate of the left eye had almost disappeared and the pupil was evenly dilated. The vitreous was still cloudy but the details of the fundus could be indistinctly seen and she could count fingers 8 or 10 feet away. The right eye was painful and congested, but the pupil was clearer than it was on the previous day. Treatment continued, with exception of subconjunctival injections. Improvement continued steadily so that in a few days the pupillary exudate had disappeared, the details of the fundus could be indistinctly seen through the hazy vitreous and the vision had correspondingly increased.

March 8, 1913, pupils were evenly dilated, no visible trace of the pupil exudate, and the vitreous fairly clear, so that the details of the fundus could be easily seen. Around the posterior pole of each eye was seen a pronounced halo of shimmering radiating lines in the retina; these were not opaque, for they would change their position slightly as the light was thrown from different angles, but they were much more intense than any halo I have seen around the yellow spot or in a "watered silk" appearance of the retina. No other change in the fundi.

Vision in each eye had improved to 6/10 with sph. + 1.00.

March 15, 1913:

R. V. c. Sph. + 1.00 Cyl. + .25 90=6/6—2
L. V. c. Sph. + 1.25 Cyl. + .25 90=6/6—3

The only remaining external evidence of the severe inflammation she had is a congestion of some of the larger veins of the eyeballs and this is disappearing.

The unusual recovery in this case might cause one to doubt that it was in reality an example of true metastatic ophthalmia of embolic origin, but this doubt was dispelled from my mind by the rapid onset, the severity of the inflammation and the clinical appearances.

Probably the infection was confined more particularly to the anterior part of the uveal tract, and whether due to the diphtheria bacillus or to a streptococcus, its course must have been profoundly influenced by the antitoxin or the reaction occasioned by the subconjunctival injections.

Fortunately cases of this disease are less frequent than they were before the introduction of antiseptic methods in obstetrics and surgery, for the pyemias and septicemias of preantiseptic days frequently manifested themselves by metastatic processes in various parts of the body and the eye was often affected.

I presume it is safe to say that the majority of cases occurring nowadays are cryptogenetic in character or complicate some distinct infectious disease. The disease may be unilateral or, in severe infections, bilateral, but both eyes are usually not affected simultaneously. The first symptom noticed by the patient is a blurring of the vision of one or possibly both eyes. If the physician is fortunate enough to be able to make an ophthalmoscopic examination at this time, he will notice that the vitreous is already slightly cloudy. The outlines of the optic disk will appear blurred and indistinct and the disk itself somewhat reddened. The retinal veins will be engorged, and tortuous because of edema of the retina.

There may be slight hemorrhages from the arteries or from the veins, and here and there yellowish-white plaques or masses somewhat similar to those observed in the retina in association with kidney disease or diabetes. Owing to the rapid onset, however, the opportunity of making an early ophthalmoscopic examination does not frequently offer, for within a few hours after the patient has first noticed the dimness of vision the media may have become so clouded as to make such an examination impossible.

In a day or two (or even earlier) after the first symptoms, congestion of the conjunctival and ciliary circulation will be noticed and some edema of the conjunctiva (chemosis) will be present. The iris will be sluggish to light or immobile, and an ophthalmoscopic examination will show only a faint reddish or grayish pupil reflex.

By this time the vision will have fallen so that the patient is barely able to distinguish the light. A deep-seated pain, possibly radiating to the side of the head, may be an early symptom, to increase with the progress of the disease. In certain cases, particularly of

the milder types, the pain may be insignificant or even absent entirely. As the inflammation increases the edema of the conjunctiva becomes more marked so that a fold of the membrane protrudes between the eyelids. The eyelids themselves become edematous and so swollen that it may be difficult to separate them sufficiently to view the eyeball. The tissues of the orbit are also distended with the inflammatory edema, so that the eyeball is pushed forward in a state of exophthalmus and its movement is limited.

If the case is seen for the first time in this stage, the physician may at first glance think he is dealing with a case of abscess from orbital cellulitis, but the chemosis and the impossibility of getting a red pupil-reflex with the ophthalmoscope should prevent this error. The tension of the eyeball is increased from the products of inflammation within it, and the eyeball may be exquisitely tender to pressure.

The iris becomes discolored and adherent to the lens with synechiae. Pus appears in the anterior chamber and the cornea becomes cloudy and opaque. The pain becomes intolerable, and finally the pus in the eyeball escapes through an opening in the sclera, which has softened and ruptured usually at the point of insertion of one of the extrinsic muscles; or the cornea may slough and thus allow the escape of the contents of the abscess.

With the opening of the eyeball the intense pain is relieved and the severe inflammation gradually subsides after the extrusion of the contents of the bulb. Then follows a gradual contraction of the sclerotic and what remains of the other structures, until the shrunken mass bears little resemblance to an eyeball. This is the condition of phthisis bulbi.

MILDER CASES

In milder cases the pain may be much less intense, the edema and exophthalmus much less noticeable, and the cornea and crystalline lens may remain clear so that the physician, with an ophthalmoscope, or even without it in a suitable light, may see in the vitreous chamber not far back of the lens a yellowish mass of purulent exudate. Such cases may not terminate in rupture of the globe, but as the inflammation subsides, the exudate organizes and ultimately after months or years the eyeball gradually shrinks, leaving the condition of atrophia bulbi (Case 2). The eye is totally blind and from the pupil, which is immobile, though frequently partially dilated, is given back a whitish reflex.

To such cases is given the name "pseudoglioma." Such a condition is occasionally seen in young children after some infective process, notably after meningitis, and it simulates glioma of the retina so strongly in some cases, especially when the history is indistinct, that a diagnosis is difficult.

ETIOLOGY

Metastatic ophthalmia may develop in the course of various forms of pyemia.

According to a classification of Axenfeld,[1] who in 1894 studied 166 different cases, it occurs most frequently in puerperal pyemia, 76 of his cases being of this kind. Surgical pyemia furnished 60 cases, and cryptogenetic septic pyemia 30 cases.

It may also develop in the course of various infectious diseases, such as epidemic cerebrospinal meningitis, sporadic meningitis, pneumonia, scarlet fever, measles, typhoid, diphtheria, cholera, variola. It has even been reported as occurring in mild form after furuncle from staphylococcus infection. Because of the more widespread use of antiseptic methods in midwifery and surgery, the puerperal and surgical cases are much less frequent than formerly.

In the development of metastatic ophthalmia the specific organism that excites the inflammation probably lodges in some of the narrow capillaries of the retina or chorioid included in small septic emboli. Virchow[2] was the first to discover such emboli and so to explain the metastatic nature of the disease. These small embolic masses promptly light up an inflammation of the retina which is the cause of the early disturbance of vision.

The question whether the retina or chorioid is first affected by this embolic inflammatory process is a debated one. Axenfeld holds that in bilateral cases the retina is first affected, whereas in unilateral cases the uvea is also primarily involved. This is certainly a fine distinction that it would seem difficult to prove. Certainly after the inception of the trouble, the circulation of the eye is interfered with by the formation of thrombi in both the retinal and chorioidal vessels. This explains the early appearance of the edema of the conjunctiva and the tissues surrounding the eyeball. In puerperal cases the micro-organism that most frequently causes ophthalmia is the streptococcus. These organisms cause stoppage of the fine capillaries with relative ease.

The ophthalmia begins most frequently in the first or second week after the puerperal infection, but it may come on after the patient is apparently well and as late as the seventh week after the infection. In cases in which it is so long delayed there is often ulcerative endocarditis which may account for the infection of the eye. Indeed, endocarditis occurs in more than half of the puerperal cases having a fatal termination.

According to Axenfeld's table of sixty-nine cases of metastatic ophthalmia occurring in puerperium, forty-two were bilateral; both eyes were affected either at the same time or with an interval of only two or three days. In four cases this interval was longer, in one of them the second eye being affected twenty-four days after the first.

The number of cases of metastatic ophthalmia occurring from surgical pyemia is much smaller than before the days of careful antiseptic technic. Cryptogenetic infections, however, still furnish a considerable number of these cases.

The pneumococcus, typhoid bacillus, colon bacillus and influenza bacillus, as also the *Meningococcus intracellularis* have been found in eyes affected with metastatic ophthalmia; but in cases occurring in connection with the various infectious diseases that have been mentioned before, measles, scarlet fever, diphtheria, cholera, etc., there is usually a mixed infection and probably some of the pus-forming organisms are the real cause of the suppuration in the eye.

A peculiarity in cases developing in the course of meningitis is, that they are of a much milder character than those caused by other infections. It is this class that frequently furnish in children the cases of pseudoglioma.

PROGNOSIS

An important part of the subject is the prognosis, not so much for the eye, which is usually lost, but for the life of the patient who suffers from metastatic ophthalmia. As to the eye, even if it escapes destruc-

1. Axenfeld: Arch. f. Ophth., xl, No. 3, p. 1.

2. Virchow: Ueber kapillare Embolie, Arch. f. path. Anat. u. Physiol., ix, 307.

tion from the suppuration, it is usually totally blind. A few rare cases have been reported in which a small degree of sight remained after the subsidence of the inflammation. Usually, if the patient survives, he has a shrunken, useless eyeball, which may have to be removed.

The danger of sympathetic disease of the sound eye is not so great in phthisis bulbi, that is one in which the contents of the ball have been extruded in the suppuration, as in atrophi bulbi, in which the ball has not ruptured, but in which the contraction of the organized exudate causes shrinking of the bulb. These latter may be dangerous and should be removed.

As to the life of the patient, the prognosis, in case of metastatic ophthalmia occurring in any infectious disease is grave, especially so if it is bilateral. Many patients die. The mortality ranges from 21 per cent. in the unilateral to 85 per cent. or more in the bilateral cases.

In puerperal pyemia, the mortality is even higher than that given, being from 55 to 65 per cent. for the unilateral and 85 per cent. or more for the bilateral cases. In the unilateral cases death occurs in from one to eight weeks after the onset of the ophthalmia, whereas in the bilateral cases it occurs earlier, sometimes soon after the involvement of the second eye. In surgical pyemia the mortality of the bilateral cases is also great; 75 per cent. while 50 per cent. or more of the unilateral cases succumb. The mortality is less in cases in which metastasis occurs in the eye alone; but if metastatic processes exist in other organs as well it is great, even as high as 85 per cent. In cryptogenetic septic cases the mortality is about the same when the ophthalmia is bilateral, but is lower when unilateral, recorded cases showing about 83 per cent. for the former, and 33 per cent for the latter.

Metastatic ophthalmia therefore is of serious prognostic import, in any infectious process, and would seem to be an index of the general infection.

TREATMENT

Usually little can be done in the way of treatment, once the infection has occurred, except to alleviate the suffering of the patient. Hot compresses, cocain applications and leeching the temples may give some relief. When it is clear that pus is in the eyeball and that panophthalmitis is inevitable the eyeball had better be incised to allow drainage. Evisceration of the contents of the globe may be done at the same time or later, according to the condition of the patient. The question of enucleation of the shrunken eyeball will have to be considered at a later time if the patient survives.

We have reason to hope that vaccine and serum therapy, applied early enough, may be of benefit in some of these cases.

ABSTRACT OF DISCUSSION

DR. OSCAR WILKINSON, Washington, D. C.: In regard to Dr. Wilder's third case, while there is little doubt that it was a case of metastatic ophthalmia, there is grave doubt that it was due to streptococcus, for a well-developed streptococcus infection in the vitreous would undoubtedly lead to blindness. The inflammation in the vitreous was possibly due to the toxin from the diphtheria. Pus-forming germs would not ordinarily leave an eye in a healthy condition when they had been sufficiently numerous and well organized to obscure vision. I doubt seriously that any pyogenic metastatic eye ever recovers after vision has been reduced to the perception of light. The inflammation from the meningococcus and several other of the milder and less active bacteria would not be likely to cause so much damage to the eye. It is a well-known fact that metastatic ophthalmia is a grave symptom usually found in cases of general pyemia, and as pointed out by Dr. Wilder, when both eyes are affected, the prognosis is ominous. It is quite probable that we sometimes have slight inflammations of the chorioid which are not sufficiently developed to attract attention, owing to the serious illness of the patient at the time, and it is possible that even pyogenic organisms might be thrown into a vessel of the chorioid without developing into a serious abscess. When this class of bacteria develop sufficiently to cause blindness, we would have grave doubts of the recovery of the vision. We are indebted to aseptic surgery and to aseptic accouchement for the decline in the number of these cases. By properly attacking the foci of infection and eradicating this, we are enabled to prevent a large majority of these cases, and a fatal termination which was formerly so frequently associated with puerperal conditions, and particularly those cases in which metastatic ophthalmia was present. The fact that metastatic ophthalmia is associated with some general infection shows that our first duty is to find the cause of the infection and relieve that as soon as possible, either by surgery or serums, or by both. Serums and vaccines really offer the only hope in the way of treatment, outside of combating the general conditions with tonics, and attacking the foci of infection with the knife and antiseptics. I regret that Dr. Wilder did not go into more detail regarding this form of treatment and its application and usefulness. The use of the proper serum is indicated in all cases of metastatic ophthalmia where bacteria are found in the blood, or even in cases in which there are no bacteria found, where there is a focus of infection as in the second case discussed. Sufficiently large doses given intravenously as the improvement or condition of the patient might indicate would no doubt prevent the fatal termination in some of these cases, even though the eyes might be lost. In cases which are sufficiently chronic, the use of both serums and vaccines is advisable. Vaccines are effective as a preventative, when an early diagnosis of the general infection which caused the metastatic ophthalmia has been made, and by its use, it is possible to prevent many of these cases. While it may not always be necessary to use autogenous vaccine, which is usually the most effective, it is necessary to discover the bacteria at fault and to administer a vaccine accordingly. If seen early enough, in mild cases, it is possible to prevent the disease, particularly if the doses given are large enough. The weakest point in the vaccine and serum therapy is the timidity with which it is given.

DR. ALLEN GREENWOOD, Boston: About five years ago there occurred in Boston an extremely severe epidemic of cerebrospinal meningitis, which increased in severity as the cases became more frequent, and then subsided, so that during the whole year we had a great many cases at the Boston City Hospital. Dr. Wilder speaks of meningitis causing metastatic ophthalmia of mild character. This may be true, perhaps, of some forms of meningitis in children. In the epidemic in Boston it was not true. The cases of metastatic ophthalmia were extremely severe. Most of the patients with metastatic ophthalmia died, but the severity was marked. One man recovered, with a complete phthisis bulbi in the involved eye. At that time Dr. Councilman called attention to the great prevalence of optic neuritis and chorioidal trouble in meningitis, and practically every patient who entered the hospital with cerebrospinal meningitis showed disturbance of the optic nerves. There is a question, perhaps, whether these cases should be called metastatic ophthalmia, or whether there was a direct extension of the infection from the fluid of the sheath, into the eye itself. In the cases I saw, the eyes were lost quickly and with one exception the patients who showed ophthalmia died from the cerebrospinal meningitis.

DR. CASEY WOOD, Chicago: In January, 1894, I was consulted by a woman who exhibited one of the rare cases of metastatic ophthalmia just referred to by Dr. Wilder, in which, although there was a bilateral, septic panophthalmitis and vision was completely lost in both eyes, the patient survived. She was 34 years of age and apparently free from organic

disease. Of her four pregnancies, only one had a living issue, a child in poor health. Of three other conceptions one ended in a miscarriage and two others in still-born children, each at about the seventh month. The first of these premature labors was accompanied by an albuminuria and followed by a mild attack of puerperal fever. Three days before the termination (at seven months) of her last pregnancy, she was seized, on retiring, by a violent headache which continued during the night and was accompanied by "cracking" noises on the left side of the head and face. When morning came, the patient could not see out of the left eye, and the lids were much swollen. During the day the eyelids on the right side began to swell, and later on, the vision was also extinguished in the right eye. The edema of the parts increased until the eyeballs protruded between the enormously swollen lids. When I saw her six weeks after the trouble began, the patient was still weak and had a rapid pulse, although her temperature was normal and she had no albuminuria. There were no lesions of the heart, lungs or reproductive apparatus discoverable on an examination by a competent surgeon. The patient presented an ocular picture that I have never forgotten. The skin about the whole orbital region on both sides was enormously swollen and thickened, the lower lids were everted and could with difficulty be replaced, while the conjunctiva, both ocular and palpebral, edematous and covered with pus, hung in folds over the margins of the lids. The cornea had almost entirely sloughed off, and from the gaping openings the pus was still oozing. Some attempt at repair had been made on the right side, where lens matter was still adherent to some partially organized scar tissue. A careful examination of the case disclosed no septic foci except those established by the puerperal state in a probably luetic subject; hence, I think we were justified in regarding the process as a bilateral panophthalmitis due to a septic embolism of uterine origin. A year after the accident the patient was still living; in fair health, but with much shrunken eyeballs. This case was reported, both in the *American Journal of Obstetrics*, 1894, and in the *Philadelphia Medical News*, April 27, 1895.

DR. EDWARD JACKSON, Denver: I recall the cases of two women suffering from panophthalmitis of one eye due to pneumonia. In one case the eye was left blind, but not much shrunken. There was no bacteriologic evidence. The other patient came into the hospital for a comparatively light pneumonia, with slight pleurisy. About the third day, the eye became severely inflamed, and burst. I think, on the third day of the ocular inflammation. The pus from the interior of the eye was loaded with influenza bacilli in pure culture. The patient then did well in a general way for ten days, at the end of which time she developed an acute endocarditis and died after being in the hospital about three weeks.

DR. ADOLF ALT, St. Louis: I agree that in cerebrospinal meningitis the infectious process simply creeps down into the eye by way of the intervaginal space. I have had a few cases in which I found this to be true. We are not dealing with a metastatic affection, but with an extension of the meningitic process through the intervaginal space down into the eye.

DR. W. H. WILDER, Chicago: I do not wish to give the impression that, because the streptococcus was found in the tonsil of the patient that recovered, therefore it was responsible for the inflammation in that eye. The presumption is that in this last case the infective agent was really the diphtheria bacillus, and that probably the prompt administration of antitoxin had something to do with checking the process in the eye. If this is true, and it was really a case of metastatic ophthalmia, which was affected by the prompt administration of antitoxin, it holds out the hope that, if we can find the organism causing the trouble and administer the proper serum or vaccine, we may hope to avert some of these disastrous cases and conserve a degree of sight. That this third case may have been due to infection with the diphtheria bacillus is not unreasonable. We can do nothing but presume in this case, because we could not examine the media of the eye for the organism, but the case presented all the clinical features of metastatic ophthalmia.

PHLYCTENULAR OPHTHALMIA AND EPISCLERITIS *

A STUDY OF THE BEARING OF THE NEWER RESEARCH ON THEIR ETIOLOGY AS THE BASIS OF A SCIENTIFIC THERAPY

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Primarily, two views are possible as to the etiology of these lesions.

A. They may be dependent on infection from without—ectogenous bacterial invasion.

B. They may be endogenous. As to the endogenous theory, several possibilities obtain:

1. They may be malnutritional in origin or due to autointoxication.
2. They may be endogenous infections.
3. They may be irritative effects of bacillary fragments.
4. They are possibly the result of anaphylactic reactions.

ECTOGENOUS THEORY

It may scarcely be claimed by students of the subject that these are diseases of bacterial invasion from without. Bacteriology has failed to disclose any consistent evidence of such invasion, and pathologic histology shows no break in the conjunctiva or site of ingress of the infector primarily.[1]

It is of interest incidentally that observers have found the histology identical as between episcleral and phlyctenular nodules,[1] namely, a conical area of round-cell infiltration. These nodules resemble but are not identical with tubercle. Of course, they are not vesicular, as the name implies. The pathology presents nothing akin to the herpetic eruption claimed by some as its prototype.[2]

ENDOGENOUS THEORY

One comes quickly then to the consideration of the endogenous origin of the lesions, and the nutritional point of view becomes at once prominent.

Malnutrition Theory.—While simple malnutrition may be shown to be a primary factor in lowering natural resistance to infection, it seems hardly proper with our present knowledge to consider it a primary cause of lesions of this type. This may be shown later.

Under this head may very properly be considered part of the most excellent and convincing contribution of Professor Straub on specific body weight in relation to scrofulosis; such a part as concerns the discussion of nutrition.

Influence of Lime Salts.—Straub[3] and his pupil, Wiemer, took the specific weight of children suffering from phlyctenular ophthalmia as compared with healthy children. They submerged these children in tepid water except for the head, measured the quantity of displaced water and calculating this and the estimated displacement of the head, determined the specific volume of the children. This table I take the liberty of introducing

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

1. Parsons: Pathology of the Eye, 1904, I, Part I; for Bacterial Observations see pp. 78, 270 *et seq.*
2. Bruns: Phlyctenular Ophthalmia and Its Etiology, THE JOURNAL A. M. A., Sept. 21, 1912, p. 1002.
3. Straub: Oxford Address, Ophthalmoscope, 1912, x, No. 11.

here, and from it will be seen at a glance that a great majority of the scrofulous children have about the specific weight of water, whereas the normal children have a higher specific weight. This lighter weight may quite justly be ascribed, as Straub claims, to the variation in the density of the bones due to deposit of the lime salts in normal children and a lack of such deposit in scrofulous types.

Exception may properly be taken to the interpretation which Straub places on these estimations, at least until such time as it may be shown whether these children may not be primarily lime-starved. It is surprising how little attention is given by practitioners, or even dietitians, to the lime-salt content of the diet in infancy and childhood.

Consideration of malnutrition is of first importance, since association of it with phlyctenular ophthalmia is at least always evidenced. Though fundamental, it cannot be the only point of view, since most children with malnutrition have not phlyctenular ophthalmia, even though the contrary association may be shown.

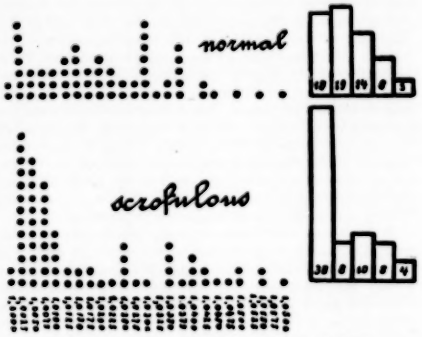

Chart 1.—Specific weight of scrofulous children.

Auto-Intoxication Theory.—So-called auto-intoxication, more properly enterotoxemia, enters into the discussion of malnutrition. The researches of Cannon[12] on the mechanics of digestion, by the combined bismuth meal and Roentgen ray, have revolutionized the ideas of scientists on this form of auto-intoxication and place it definitely under the head of a stasis. As now proved enterotoxemia is due, not to infections of the mucosa *per se*, but to retention of the materials by stasis, the incubation of a polybacterial flora leading to the formation of many varieties of poisons. Further consideration of this will be taken up specifically in the discussion of the anaphylaxis hypothesis.

Notwithstanding the fact that these points with reference to the nutritional side are fundamental, they may more properly be classed as predisposing causes, for it has never been proved that a definite lesion, such as we have under consideration, could be produced primarily by errors of nutrition. Something else is necessary, something of an infectious or specifically irritating nature. And so we come to endogenous infections.

Endogenous Infection Theory.—So the puzzling nature of the question has led, as you know, to the assumption of metastases from some hidden focus in the body, and Straub takes such a latent tuberculosis as the morphologic substratum of scrofulosis, the result of a preinfection with tubercle bacilli, unhealed and still containing living bacilli. The benign nature of this metastatic scrofulosis is taken to be due to an attenuation of the bacilli combined with a partial bacterial immunity which has been developed through the preinfection, one or both. These lesions do not develop into genuine tuberculosis because either the bacillus is attenuated or the spontaneous immunity is controlling. On this theory there must, accordingly, be many grades of virulence of the organism and much variation in the antibody formation—endless shades of toxic and infective and antibody potency.

This lack of development of full tuberculous lesions secondarily was the fundamental discovery of Koch, who undertook superinfection on guinea-pigs as long ago as 1891, but produced only quick-healing ulcerations without even involvement of the neighboring lymph-nodes. Hamburger[13] has followed this out further and concluded that it is a sign of immunity dependent on hypersensitiveness, or allergia, the organism immediately repelling the infection by the quick reaction of "natural inflammatory healing forces." Römer[14] has carried us still further and proved that even this second infection does little to extend the primary lesion.

Two possibilities are offered to explain the negative finding of tubercle bacilli in these lesions: (1) imperfection in staining technic; (2) their quick destruction after infecting. Quite recently Wegelin,[15] in a study of supposed healed calcareous tuberculous lesions, was able to demonstrate tubercle bacilli of low virulence by a special process of staining, namely, the alkaline hypochlorite method, so that it remains to be seen whether one might more often demonstrate tubercle bacilli in these lesions by employing more appropriate methods of staining. The conclusion is also justified by this and by the work of Rabinowitsch and Schmitz that the tubercle bacilli in most of the so-called healed tuberculous lesions, even the calcareous or encapsulated ones, are still viable.

Bacillary Fragment Theory.—Klingemüller[17] has claimed that these tuberculids, which might be taken to be akin to phlyctenas, are the result purely of toxic actions, and he cites the fact that tuberculin (which is the protein of dead tubercle bacilli) may be filtered through Berkefeld filters and, although assumed to contain no bacillary particles in consequence, the filtrate will still produce not only tuberculids but true tuberculous tissue. Siegrist[18] has produced anatomic tubercles by the conjunctival reaction of tuberculin.

In the early experiments the tuberculins employed were not true solutions and hence not free from fragments of tubercle bacilli. Previously it had been many times proved that pulverized tubercle bacilli could produce miliary tuberculosis, and the tubercles were thought by Pick and Daels to be due to bacillary fragments. Zieler[19] later clearly showed that these tubercles could be produced not only by filtered-out tuberculins, from which

12. Cannon, W. B.: The Mechanical Factors of Digestion, 1911.

13. Hamburger: Beitr. z. Klin. d. Tuberk. (Brauer's), 1909, xii; section 3, Ueber Tuberkuloseimmunität.

14. Römer: Beitr. z. Klin. d. Tuberk. (Brauer's), 1908, xi, section 2, p. 109.

15. Wegelin: Cor.-Bl. f. schweiz. Aerzte, 1910, xl, 913.

17. Klingemüller: In Wolff-Eisner's Frühdiagnose und Tuberkuloseimmunität, 1909, ed. 2, p. 130.

18. Siegrist: Therap. Monatsh., April, 1908.

19. Zieler: München. med. Wchnschr., 1908, No. 32, p. 1685.

it was assumed that the microscopic fragments were entirely absent, but even by dialyzed tuberculin, which would supposedly contain only the dissolved substance.

Especially is attention directed here to the so-called micellae of Nägeli—for the ultramicroscope reveals these dialyzed tuberculins as molecular aggregates. These micellae are in harmony with the accepted chemistry of colloids, for even in the dialyzate of colloids[20] ultramicroscopic bodies are present. It becomes a matter then merely of relative fineness of molecular aggregates. In this way have been explained the exanthems which we so often see after the administration of serums,[21] the minute ultramicroscopic aggregates of the proteins or albumins of alien serums being deposited in the form of metastases over the body.

Possibility of Other Organisms.—If then the production of lesions such as phlyctenas may be explained and proved possible by even ultramicroscopic metastases of bacillary protein from tubercle bacilli, may the same be proved for other organisms? On this one may only predict from still unfinished observations; similar possibilities may be shown for the bacillary protein of streptococcus, perhaps for others also.

Anaphylaxis Theory.—I take up briefly, then, the last hypothesis offered to explain the existence of these lesions, that is, that they are due to an anaphylaxis.[22] Any anaphylactic—better allergic—reaction depends on presensitization and, in this instance, we may say also presensitization of an area. An anaphylaxis hypothesis, therefore, does not explain primary causation, but is of infinite importance in the production and recurrence of the lesion after infection, as shall presently be shown. This seems to be the point of contact by which this theory may be linked with the others to complete the etiologic chain.

The study of tuberculosis must first be made because of the greater probability of its etiologic importance through force of its extreme prevalence, a point which is absolutely fundamental to the discussion.

The studies of Nägeli[23] and Burckhardt[24] show[25] that practically the whole race has tuberculosis in an active or latent or cured state, that practically all children gain this infection before the age of 12 to 15. But as the mortality ratio is probably not more than 15 per cent., it is obvious that at least a relative immunity is obtained in the other 85 per cent. This relative immuaity is indicative either of the low or diminishing aggressivity of the bacillus, or of the easy though slow evolution of antibodies, or both.

It seems, then, that at least preinfection with tubercle bacilli is capable, as is tuberculin in like manner (Case 1), of generating such a lesion as a phlyctena or an episcleritis, and one would seem justified in stating that by similar means a streptococcus would cause such a lesion as episcleritis. But it does not seem that such a lesion could be produced primarily by malnutrition or auto-intoxication independent of a preinfection.

Acknowledging that tuberculosis would most likely be grafted on a subject of malnutrition or of auto-intoxication, in what way may the problem differ when we have malnutrition or auto-intoxication subsequent to the evolution of a tuberculous focus? Here enters the influence of allergic reaction, and its study should help clear the field, and should satisfy the claims of the believers in the anaphylactic nature of such diseases; for while products of vicious metabolism or auto-intoxication may not cause lesions, their influence on the reactivation of infected foci is more open to proof.

Our point of view must, therefore, be changed in the light of these modern conceptions,[27] for they are in harmony not only with accepted theories of allergic reactions but fulfil the proved doctrines of the side-chain theory of Ehrlich and add further proof of the interrelation or similarity of hypersensitization and immunity.

So also must be weighed the importance of recognizing the local nature of this sensitization, in addition to its general character. These areas, once involved, are supersensitized and become the most likely areas for involvement; hence recurrences are prone to become manifest.[28] Certain of these substances are sufficiently attuned to the endotoxins of the infecting bacteria to act as they would in the blood-stream, and by irritation of the foci may lead to reactivation. This I take as the chemopathologic substratum of the allergic or anaphylactic reaction, the cause of the lighting up of the phlycten, or the episcleritis, and the explanation of the bearing of anaphylaxis on the etiologic problem. These substances operating by reactivation of a preinfection may excite further metastases, but they may not primarily cause the lesions, since they are not themselves specifically infective. In this way may be explained the idiosyncrasies to strawberries (Case 2) and other organic acids, the autogenic toxins involved in fatigue states, those of enteric origin, and all the array of causes that have hitherto been thought to influence and which always accompany these ocular manifestations.

Determination of Cause.—In the determination of the probable fundamental factor in a given case before us we have first to consider, because of its universal prevalence, the probability of an underlying tuberculosis. It is the great possibility of variation, not only in the attenuation of the infector but in the variability of the antibacterial immunity present, which makes the problem so complex. Studies of the opsonic index have been made and negative phases to tuberculosis have been found to precede the evolution of a phlyctena. This may be accounted for by auto-inoculation from a reactivated focus. The appearance of the phlycten thus might properly be termed an "auto-Calmette." An opsonic observation of many cases, however, has convinced me that this negative phase is not present in the majority of cases, and obviously would only occur in cases in which the auto-inoculation was considerable. In the determination of the presence or absence of scrofulosis it would seem, as I have pointed out before—the result of frequent observation—that a variable opsonic index, not as to whether it is low, but whether it is greatly variable, points to the occasional auto-inoculation and the presence of an active focus somewhere in the body (Case 3). I take this to be a better barometer than a continuously low index.

It must be apparent to an unbiased observer that no advantages accrue from a large diagnostic dose over a

20. Ricketts and Dick: Infection, Immunity and Serum Therapy, Chicago, American Medical Association, 1911, p. 243.
21. Sahli: Tuberculin Treatment, 1912, p. 115.
22. Verhoeff: Internat. Cong. on Tuberculosis, Washington, Sept. 28, 1908.
23. Nägeli: Ueber die Häufigkeit, Lokalisation und Ausheilung der Tuberkulose, Virchow's Arch. f. path. Anat., 1900, clx, 426.
24. Burckhardt: Ztschr. f. Hyg., 1906, lvi, 139.
25. Autopsies of our cities when carefully made with that intent disclose confirming evidence of these researches.
27. The speculative nature of these reactions must of course be acknowledged. The problem is most complex because the chemistry of these bodies is not known. For the present its solution rests largely on physiopathologic reactions. Certain conclusions are, however, justified by these studies, pending chemical verification.
28. In the exhibition of several hundred therapeutic inoculations of many vaccines I have satisfied myself that the areas of inoculation remain sensitized for a considerable period of time and many times become irritated when reinoculation is employed in other regions. This to my mind is akin in a small way to what happens to the phlyctenular area.

minute dose therapeutically administered but giving a diagnostic reaction; for a therapeutic administration of tuberculin which has produced a reaction has established a diagnosis of sensitiveness, and that is the aim of the test. Such a diagnostic measure may indeed serve both for diagnosis and for control of therapy, a guide to subsequent administration. The second test of reaction, and one which Sahli recommends, is the examination of the leukocyte count for an increase (Case 4). The leukocyte reaction is thought to offer the mildest reaction possible and is the one which I employ for therapeutic guidance.

I accept the Wolff-Eisner theory of lysin formation as the cause of the tuberculin reaction, and the fact that an autogenic immunity is adequate to preserve life in 85 per cent. or more cases shows at least that a sustained resistance is established, a condition I propose to call "defensive equilibrium." Now, Wolff-Eisner assumes the existence in the tuberculous infected macro-organism of amboceptors or lytic bodies, and bases his theory on the well-known allergic reactions which follow the injection of albuminous substances, parentally, namely, the formation of albumin-dissolving substances, albuminolysins. After the first injection they are present in the circulation and the macro-organism is sensitized to subsequent injections. These lysins are formed in excess of the requirement of the first injection. (See Vaughan and Wheeler's theory also[31].)

When tuberculin is inoculated in a case in which there has been a preinfection with tuberculosis, there is a speedy cleavage due to the presence of the lysin, the formation of tuberculinolysin, which itself forms, first, primary lytic antibodies, leading to attenuation and destruction of bacilli acting directly (bacteriolysins), then secondary antitoxic antibodies which neutralize the toxicity of the lysinized tuberculin, as well as the lysinized tubercle bacilli themselves.

Tuberculin (injected) + lysin (already in body) = first, the formation of tuberculinolysin, increased toxicity, leading secondarily and later to the formation of antibodies antitoxic thereto.

Now, if body-made bacterial protein produces such manifestations as phlyctenas and episcleritis, why does not ectogenic tuberculin produce the same manifestations? The answer contains both the proof of the Wolff-Eisner theory and the strongest possible argument for small dosage. An irritation shown in the foci or in the production of a phlyctena not only furnishes the proof of the Wolff-Eisner theory but shows that the dosage has been too large, has been greater than the serum content of antibodies can control, has done temporary harm.

Hence, since the toxin formed by the tuberculinolysin is the cause of the reaction, any excess of tuberculin would produce a reaction and that reaction would evidence lack of antitoxic antibodies in the serum. For that reason, as will be stated in connection with the therapy, only reactionless therapy may safely be employed.

THERAPY

General Therapy.—Prof. William F. Norris inherited from Von Graefe the treatment of phlyctenular ophthalmia[32] which was employed in his service at Wills Eye Hospital in the early days of American ophthalmology. This treatment is still in vogue in the clinic of his successor and is a beneficent example of empiricism in the specialty. It was based on careful observation of therapeutic effects. I have seen too many patients cured by it to believe that it was anything but scientific, or to minimize the importance of the local, general and gastro-intestinal therapy. It is not now out of line with scientific findings, albeit its advocates could not have appreciated its scientific foundation.

The treatment consisted in the withdrawal of all cane-sugar combinations, all acids, tea and coffee, and the sterilization of the intestinal tract by absorbing doses of calomel continued over many days; they substituted fresh air, nourishing, bland diet—a good basis for the natural immunizing treatment of to-day.

My therapy since these days of tutelage has been this treatment amplified by the early realization that what must be sought is what we have always termed an alkaline dietary. To that end for twenty years my practice has tended toward lower and lower protein and a non-acid[33] content of the diet. Latterly has been proved the necessity for protein in the anaphylotoxin from which the allergia are produced. The value of this low nitrogen intake has had latter-day confirmation by scientific researches on nutrition, as Chittenden[34] on the physiologic, and Metchnikoff and Combe[35] and many others on the auto-intoxication side. Further help has been offered by attention to the lime content. Improved, too, have been the means of gastro-intestinal sterilization, speaking now from the prophylactic side in the management of these sensitized subjects. These have been afforded by the work of Combe,[35] Metchnikoff, Von Noorden,[36] and the classical researches of Cannon,[12] above mentioned.

Specific Therapy.—Quite compatible with this general management, but far more fundamental for the immediate attack, is the specific therapy, and with no disparagement of the former it must be said that the latter justifies itself in the immensely shortened duration of the attack (Case 5, also other appended cases).

Freed from the empirical, there should be no hesitation in the immediate application of both this general and the specific therapy to the case in hand. One who does not appreciate the fundaments and clearly comprehend the theories of specific reactions should not undertake the use of immunization treatment in any department.

Reactionless Therapy.—In the beginning of the consideration of specific therapy, which may only be touched, I accept the dictum of Sahli, of Wolff-Eisner, and of Wright, evidenced by their many writings, for small dosage—for reactionless therapy. Wright has led in this practice, and one finds in the successful therapeutic inoculation methods of Wolff-Eisner and Sahli an extension of Wright's valuable work.

I confess that one has great difficulty in restraining oneself from obtaining a grossly visible reaction in a therapy of this type, but one is repaid for such restraint by the valued and many times ideal results. There is small solace for the physician in seeing a reaction if the patient is thereby made worse.

Many precautions are necessary for successful employment of tuberculin, but into these I may not go. In my observations all grades of reaction have been manifested—local alone, focal alone, or general alone, or two, or altogether, or all absent with only the white cells increased; and this great variability in the reaction, not

31. Vaughan and Wheeler: Jour. of Infect. Dis., June, 1907.
32. Norris and Oliver: Text-Book of Ophthalmology, 1893, p. 328.

33. It is not generally known that subjects of enterotoxemia are idiosyncratic to organic acids of all kinds, but such is the case, though the reason for it is not clear.
34. Chittenden: The Nutrition of Man, 1907.
35. Combe: Intestinal Auto-Intoxication, 1908.
36. Von Noorden: Intoxikation Proceeding from the Intestine, THE JOURNAL A. M. A., Jan. 11, 1913, p. 101.

only as between patients, but on different occasions with the same patient, would seem to indicate the great variation in lysin activity or complement, possibly a rise and fall in them, as has been shown in so many other bodily functions.

Other, better controls of this therapy than the white

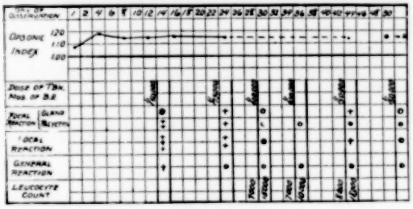

Chart 2.—Opsonic, leukocyte and other reactions described in Case 1. In this and the following illustrations ⊙=doubtful, +=positive and 0=negative.

blood-count may be forthcoming, but we should at present be content to produce increase in the leukocytes in twenty-four hours to the exclusion of other reactions. This satisfies the call for evidence of effect, for the present at least.

One who adopts the low dosage can help all cases which may be helped by the therapy, while the large dosage is two-edged and will help a minority to the injury of many subjects.

Sahli has well summed up the question when he says that the effect of physiologic irritation is not generally proportional to the strength of the stimulus but rather depends on a condition of the organ stimulated. Thus, a weaker stimulus often produces a greater effect than a stronger, since the latter may paralyze the function. Confirmatory evidence of this is found in the work of Friedberger and Moreschi.[38]

REPORT OF OBSERVATIONS

CASE 1.—Demonstrates occurrence of phlyctena on overdose of tuberculin administered for mastoid pain due to deep subauricular lymph-node involvement. Chart 2 shows also opsonic, leukocyte and other reactions. Patient was a woman aged 46 years. History later disclosed fact of frequent occurrence of phlyctena years previously. Note lack of variation in opsonic index of case which, nevertheless, responded to tuberculin. Also leukocyte and other reactions quite marked. Minute doses employed.

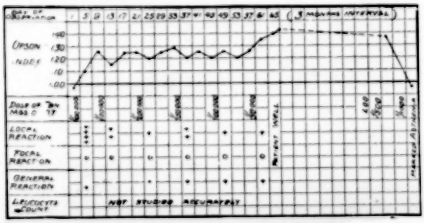

Chart 3.—Variable preinoculation opsonic index described in Case 3.

CASE 2.—Man of 26 years. Demonstrates occurrence of phlyctena on ingestion of unripe strawberries. Cure by reactionless tuberculin in five days. Patient has observed frequent association of phlyctena with eating of strawberries. Other attacks have lasted two and three weeks. Questionable history of early tuberculosis. Blood not studied.

Dosage first day 1/100 mg., fourth day 1/50 mg. B. E.

CASE 3.—Demonstrates variable preinoculation opsonic index in the case of episcleritis cured by tuberculin after all other therapy had been tried and failed. Patient a robust man of

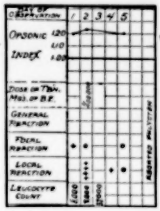

Chart 4.—High opsonic index associated with other marked reactions in abortive case of phlyctena described in Case 4.

48. Case diagnosed as of probably rheumatic origin and so treated. Seen by Wilder of Chicago and Wilmer of Washington who concurred in diagnosis and treatment. Treatment failed. After seven weeks vision reduced to fingers at 2 feet by cloudy punctate cornea. Blood studied and cure effected and vision restored in twenty-one days by reactionless therapy. Chart 3, opsonic index and other findings. Response to tuberculin immediate.

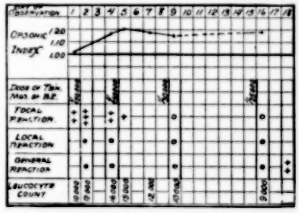

Chart 5.—Quick recovery from episcleritis described in Case 5.

CASE 4.—Demonstrates marked leukocyte reaction in abortive case of phlyctena, also marked local and general reactions. Patient girl of 17 years, sister of Patient 2. Later learned history of preinfection. Pott's disease in early childhood. Chart 4 shows high opsonic index associated with other marked reactions.

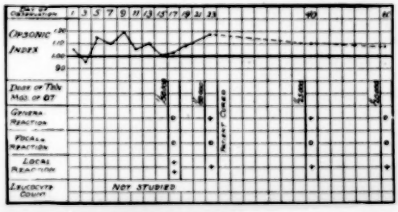

Chart 6.—Note association of raised opsonic curve with improvement, and drop after diagnostic dose.

CASE 5.—Demonstrates quick recovery in case of episcleritis from reactionless tuberculin therapy. Man of 42 years. Had many previous attacks of episcleritis which were invariably protracted. Chart 5 illustrates this case.

CASE 6.—Demonstrates extreme case of scrofulosis. Girl aged 20. Recurring phlyctenas complicated with deep peri-

38. Friedberger and Moreschi: Deutsch. med. Wchnschr., 1906, No. 49, p. 1986

osteal ulcer over mastoid. Mother died of phthisis when patient was born. Steady improvement under reactionless tuberculin therapy including healing of ulcer and improved general health. Chart 6 shows association of raised opsonic curve with improvement and drop after diagnostic dose. After two months steady improvement usual diagnostic dose administered by practitioner followed by two usual therapeutic doses leading to perilous asthenia — marked allergic reaction.

Collectively these observations show the inconsistency of the reactions to which the tuberculous patient is liable, both from his disease and from tuberculin inoculation. Any one of these may be considered a positive reaction since none of them should show in the non-infected. These cases indicate also the possibility of inducing the reactions with minute doses and seem to substantiate the argument for the sufficiency of the employment of such therapeutic inoculations for diagnostic purposes as well.

ABSTRACT OF DISCUSSION

DR. GEORGE S. DERBY, Boston: More and more evidence is being presented each year in regard to the etiology of phlyctenular disease and episcleritis which shows that these cases rest on a tuberculous basis. Ninety per cent. of the cases of phlyctenular disease give a positive tuberculin reaction, and 10 per cent. is a small percentage of error for any test of that sort. We do not know why this percentage do not react, but I feel sure probably all of them are tuberculous in spite of the fact that they do not react. A positive reaction to tuberculin shows that the patient is suffering from tuberculosis, while a negative reaction is not so important. If the general physical examination of these patients gives a negative report, it means absolutely nothing. There are often healed lesions of tuberculosis which can not be found. Of course a positive report is good evidence. If phlyctenular disease of the eye rests on a tuberculous basis, it places a large load of responsibility on the ophthalmologist, because it is one of the most frequent diseases we meet in our clinics. It means that each year we see many children who have been comparatively recently infected with tuberculosis, and any tuberculosis expert will tell you that is the time to take tuberculosis patients—when they are young—and it is our duty to take hold of them and follow them up and see that they receive proper, general hygienic treatment; see that they are taught to live properly, and if this is done, they will disseminate the information among others in the community. By following up the cases we shall find active tuberculosis in their families. I have seen children with phlyctenular disease in the hospital, and sending a nurse to the home, found a patient there with active tuberculosis. If the ophthalmologist takes these cases up in the way he should he can aid greatly in the fight against tuberculosis. We are also aiding in the conservation of vision, because, although few of these patients actually lose their vision as the result of phlyctenular disease, nevertheless, frequent attacks are apt to damage the vision and disable the patient industrially to a considerable extent. Every hospital should have some sort of special organization to take care of the cases of phlyctenular disease. I think that they are best taken care of by means of the group method; and one cannot handle them without some sort of a follow-up system, such as a social service department, by means of which these patients can be followed to their homes, and correction made of the faults of living that are found there. It is easy to cure a single attack, but to prevent future attacks is the problem, and that is a much harder one. The use of tuberculin in phlyctenular disease is not often necessary. Most of these patients recover without tuberculin. Even though tuberculin is used, it is often extremely difficult to prevent repeated attacks in obstinate cases. We use tuberculin in Boston in a number of cases, but not in the majority. The use of tuberculin is not all that is necessary; it is not nearly so important as general hygiene. This is also true in tuberculosis in adults. I have used calcium lactate and various dietetic treatments in phlyctenular disease, and have not been very well satisfied, for I have not been able to draw any conclusions from their use. Small doses of tuberculin are preferable in severe ocular tuberculosis because it is not safe to get a reaction or to just fall short of getting a reaction. I have seen one patient treated with large injections, with the purpose of obtaining a reaction and that patient is blind to-day as the result, I believe, of the repeated reactions. Dr. Gamble is quite right in what he says. Many of the patients will recover whether they are treated or not. It is better to inject the tuberculin between the shoulder blades or sometimes in the arm. In cases of special sensitiveness there is one situation better than all the others, and that is described by some as the northwest corner of the buttock.

DR. CHARLES N. SPRATT, Minneapolis (exhibiting a patient with tuberculous keratoscleritis): This patient showed the characteristic tendency of the disease to relapse. She was seized with terrific pains and redness of the right eye eleven weeks before I saw her, in June, 1911, and examination showed inflammation of the sclera in the upper, outer quarter. I gave 2 mg. tuberculin (T. O.) as a diagnostic dose. This was followed thirty-six hours afterward by a local, focal and general reaction. The symptoms cleared up almost immediately after this diagnostic dose. She was under treatment about nine months, having had no pain after the first week. She has since had one or two exacerbations. I saw her a week or ten days ago for the first time in a year. The eye has reddened in the last few days as one week ago it was white. This is her menstrual period. The hot weather has irritated the eye. The cornea became involved after the sclera had apparently cleared up. There are now several small, dense leukomas with calcification and I believe that these inflamed leukomas are causing the trouble. She received, altogether, sixty-two or sixty-four doses of B. E., as we tried to avoid a reaction. The first dose was one-ten thousandth mg. and it was run up to 2 mg. The question arises as to the size of the dose producing a reaction. I talked with Dr. Hamman in Baltimore, who says it makes little difference whether we use one variety of tuberculin or another, but to avoid reactions. I have been impressed, however, by the fact that improvement immediately followed a diagnostic dose which produces a slight reaction. This has occurred in eight cases of scleritis and keratoscleritis, treated with tuberculin.

DR. ALBERT E. BULSON, JR., Fort Wayne, Ind.: I agree with what Dr. Derby has said concerning the efficacy of small doses of tuberculin, continued over a long period of time. I do not believe that we are justified in giving large doses of tuberculin for therapeutic purposes. The production of antibodies can be stimulated with small doses, and they are safer. I want to commend Dr. Davis for giving the von Pirquet test first. I do not see the necessity of subjecting the patient to the subcutaneous test, previous to the von Pirquet, though I do think that when we want definitely to decide the question, there is nothing equal to the subcutaneous test. The injections should be carried over three tests, giving a small dose first, and the last dose may be anywhere from 7 to 10 mg. to test the possibility of getting a reaction. If a reaction occurs following the first dose, it is well to wait a while and then repeat the test to verify the findings. I cannot quite agree with Dr. Derby as to the value of dietetic and hygienic treatment without the administration of tuberculin. It works well among the poor in cities, who do not have fresh air and good food, but the addition of tuberculin brings about still better results, as has been demonstrated rather conclusively at some noted tuberculosis camps. I have seen quite a number of patients with phlyctenula of the cornea who began rapidly to improve when given tuberculin, and who did not show such improvement on dietetic and hygienic regulations with the usual ocular treatment added thereto. It does not make much

difference what preparation of tuberculin is given, if small doses are used. I have used the old tuberculin and the tuberculin emulsion. At present I am using the emulsion in small, but increasing doses, and like it very much. The treatment is administered every five to seven days, and the patient is carefully watched for results. A reaction is not expected or desired, and the dose is diminished if a reaction occurs. The tuberculin treatment is omitted if the patient has more than from three-fourths to one degree of fever.

DR. S. L. LEDBETTER, Birmingham, Ala.: I learned to treat these things before we knew anything about tuberculin, but have used it a little experimentally, for diagnostic purposes. I have been treating these conditions for about thirty-three years, and never had a patient who did not get well. If they all recover without tuberculin I can not see any great necessity for using it. I can recall but two chronic cases. I know one young woman, married, with three children, whom I began treating when she was a year and a half old. This woman had a few recurrences but never had much trouble although her mother was tuberculous. The child never developed any symptoms except the phlyctenules. I only recall one patient whom I have treated who afterward died of tuberculosis. About one out of every hundred develop tuberculosis whether they have phlyctenules or not, so I would not have wondered very much if one or two patients afflicted with phlyctenules had died of tuberculosis. I have never had any great trouble in treating phlyctenula with good fresh air, good food and tonics.

DR. MARK J. SCHOENBERG, New York: We have been told that a general and focal reaction after the injection of tuberculin means tuberculosis, but we neglect to give sufficient attention to the counterpart of the question. How can we know that the case is tuberculous when the reaction is negative? I expected to hear this discussed. We know there are cases of lues which give a negative Wassermann, and also cases of tuberculosis, positive in character, which give a negative reaction to tuberculin. Is this the fault of the tuberculin or is it the fault of the patient? Very little is said in the literature about the work done with tuberculin of bovine origin, relating to the pathology of ocular tuberculosis. Schieck in 1911 published an article on tuberculin from bovine bacilli, injected into rabbits. It is much more virulent in the eyes of rabbits than the human tuberculin. Thus we have a range of virulency, and there may be a large range of virulency for human beings. I do not think this part of the subject has been covered at all. In using the intradermal injections of tuberculin for diagnostic purposes, we sometimes meet cases showing hypersensitiveness to the human tuberculin, and we have to change to the bovine, when we find there is no hypersensitiveness to this kind of tuberculin. Another point is the lack of knowledge why some cases do and some do not respond to tuberculin, although both classes of cases have tuberculosis. We know that phlyctenular keratitis and conjunctivitis may be due not only to the tubercle bacilli, but to foci of streptococci and staphylococci. One of the speakers may be right when he said that we are cured with and without tuberculin, so that it may not be due only to the tubercle bacilli. I think the main difficulty in lung tuberculosis is not a pure tuberculosis. Incipient cases are easily cured. The great difficulty arises with those cases in which added infection comes on top of the tubercle bacillus with the staphylococcus and the streptococcus, and the same may be true in ocular troubles.

DR. WALTER BAER WEIDLER, New York: One year and a half ago Dr. E. S. Thomson, who is the chairman of the scientific work at the Manhattan Eye and Ear Hospital, asked me to make a report on work done in the clinic with tuberculin. This report has been published in the report of the hospital for 1913. I was able to observe twenty or more cases, which included the following conditions: phlyctenular conjunctivitis, episcleritis and scleritis, keratitis, iridocyclitis and chorioiditis. This work has been continued in my service at the hospital and the von Pirquet test alone was depended on for our diagnostic information. Whenever the reaction was positive, we at once proceeded with the injection of tuberculin and the "T E" was always used, and in not a single case in which the von Pirquet was positive have we failed to arrest and cure the condition. The word "cure" is perhaps out of place in connection with work done with tuberculin; as I do not think that we cure such patients, as they are very likely to have relapses and to have the same conditions appear in the other eye. I have not used any other forms of general medication and the local treatment consisted of boric acid and atropin. In all of the cases outside of the hospital, no effort was made to change the diet or the hygienic conditions, as I wanted to test the value of tuberculin without any of the other usual means employed in the treatment of these conditions.

DR. MELVILLE BLACK, Denver: Since Colorado has a reputation as a health resort for tuberculous people, it may possibly be of interest to know that we see comparatively little tuberculosis of the eye, either in adults or in children. Many of the people who come to Colorado on account of tuberculosis afterward become the parents of children and it would be but natural to expect, if there is much in heredity, that these children would probably be afflicted with joint and general tuberculosis of some sort, either in early life or later. Yet I think most physicians will bear out my statement that this does not seem to be true. Of course we see tuberculosis of the eye in Colorado, and if we accept the modern investigations that phlyctenular keratitis is a manifestation of tuberculosis somewhere in the body, and that the local infection is either directly or indirectly a tuberculous process, we would naturally expect to see more phlyctenular keratitis. But we see very little, and almost invariably in the ill-fed, ill-nourished children who have not been properly taken care of from the cradle up. We do not see phlyctenular keratitis in the children of well-to-do families unless in exceptionally rare instances. I have had more or less experience in the use of tuberculin and desire to line myself up with those who are using it; I believe it is a means of arriving at a more definite, positive end than by the usual methods. I have been involved in a maze of conflicting emotions after reading the classical paper of Dr. Walters, and I am somewhat afraid that it would take about as long for me to understand his meaning as it would for the light of a star of lesser magnitude to reach the earth. In consequence, I hasten to agree with Dr Walter for fear of exposing my ignorance. I trust, however, that his sense of humor will enable him to appreciate my position, and that he will continue to knock at the doors of our understanding until we let him in.

DR. J. S. WYLER, Cincinnati: I want to agree with Dr. Schoenberg and Dr. Black in some of the statements made. I do not feel that phlyctenular conjunctivitis or keratitis is purely a tuberculous condition. We have been able to sensitize animals to tuberculosis, but the sensitization has failed to produce phlyctenular conditions in those animals. However, when we have sensitized them, then, with the assistance of the *Staphylococcus aureus* we can produce typical phlyctenules in rabbits and guinea-pigs. This goes to show that the condition is a mixed infection on a tuberculous basis. I have had quite a number of cases of well-nourished young children in good circumstances who developed phlyctenular conjunctivitis and keratitis, and who, in later years, have shown not only no pulmonary or general tuberculosis but not even a recurrence, so that I believe the essential point is a staphylococcic infection on a possible basis of tuberculous origin that causes this condition. Hence I feel justified in condemning the use of tuberculin in this disease, feeling sure that less radical measures produce as good results.

DR. J. A. DONOVAN, Butte, Mont.: Until I found out otherwise, I agreed to some extent with Dr. Ledbetter that many or

all of these patients would get well. In one case, a man had relapses five different times. I put him on tuberculin treatment after I had exhausted the materia medica and he recovered. Following that case, a patient was sent in to have the eye enucleated. Having obtained such good results in the former case with tuberculin, I used it, and much to my surprise she regained vision to the extent of 50 per cent. and now has a good eye. I have treated six cases to completion, and have quite a number of cases under treatment by physicians in different places. These are the cases that I have had trouble with. It is pretty hard to explain to some one else what you want done and what you mean by reaction and dosage. We talk about small doses and large doses, and I do not know what you mean by small doses, and you do not know what I mean. I have started with the fifth dilution of an imported tuberculin (Koch) which makes about 1 drop of tuberculin in a gallon and a half of water, injecting 3 minims. If I do not get a reaction, I inject 5 minims. If I obtain a reaction, when it begins to subside, after from three to five days, I give a second dose. If I have quite a reaction, I repeat the same dose or give a smaller one. If I get little reaction, I add 2 minims to the dose. Formerly, I waited until the patient was normal before giving the second dose, but I read somewhere that better results were obtained by following it up more quickly. In my early experience I got considerable reaction; two were alarming. I changed from one preparation to another, giving the same dose, not knowing that the two preparations were not standardized the same, and I had a fearful reaction. Fortunately the patient recovered.

DR. HIRAM WOODS, Baltimore: I want to follow up a remark made by Dr. Donovan, that the patient became suddenly blind from too great reaction. I have had a dread of local reaction in chorioiditis because there is not much room for such things if the exudate is near the fovea. Last winter I saw a woman, aged 37, married, and the mother of healthy children. One eye showed central chorioidal atrophy with practically no vision. The other eye had 20/15 central vision, good fields and accommodation, but there were peripheral chorioidal scars. The disk of this eye was red, not edematous, but the sort of disk which makes one suspect chorioidal hyperemia. An internist thought the urine indicated intestinal intoxication, but that a Wassermann, and, if this proved negative, a tuberculin test were indicated. The former was negative. Temperature was taken for two or three days and then 1 mg. of old tuberculin was used. There was no systemic reaction at all, but after three or four days the good eye showed cloudy vitreous, a perifoveal chorioidal exudate developed and vision fell to 10/200. Up to a month ago it had not improved. A day before the new chorioidal trouble was noted, the pathologist made a luetin test with a preparation received that day from the Rockefeller Institute. There was violent local reaction with formation of a necrotic ulcer on the arm which healed only after several weeks. Two interesting points are presented. The first is positive luetin reaction with negative Wassermann and no history even suggesting syphilis. Why was one negative and the other positive, and did the positive reaction mean syphilis? The second point, which is of great importance at the moment, is: Was the woman tuberculous without other than ocular symptoms, and has her eye suffered from a diagnostic zeal well meant, but possibly excessive? We accepted the indications from the luetin test and have treated her with mercury. Yet I am not so sure as I would like to be that we did not stir up a trouble in the good eye which might have stayed latent for an indefinite period.

DR. WILLIAM ZENTMAYER, Philadelphia: Dr. Walter speaks of the treatment pursued by Dr. Norris, my predecessor at Wills Hospital. He does not state what it was. The internal treatment was fractional doses of calomel three times a day and certain directions in regard to regime. The child was to have milk, eggs and fresh meat, plenty of fresh air and no sweet things and no fried food. He admits its efficacy and gives the explanation for it.

DR. W. H. WILDER, Chicago: There is one phase of the treatment of these phlyctenular cases that cannot be too strongly emphasized, and that is the importance of fresh air. This was particularly impressed on me last year when visiting the late Professor Eversbush of Munich, who was much interested in the subject of the treatment of scrofulous or tuberculous eye diseases in children, which in cities, among clinic cases, are often so obstinate. Eversbush established a sanatorium at Tolz in the Bavarian Alps, an ideal situation for the treatment of such cases, and when I visited it with him, he had about thirty-five children there who were suffering from obstinate corneal or uveal lesions of a tuberculous type. They were being greatly benefited by the fresh air, in conjunction with the regular local treatment, administered by the nurses and physician in attendance. He had observed excellent results from the treatment of such cases in his sanatorium, and noted that they recovered much more rapidly and with fewer complications than similar cases treated in the city. If would be well if we could get the cooperation of certain sanatoriums and give our clinic cases the benefit of fresh air, combined with measures to improve the general nutrition. Unless we get a local reaction in the eye, we are not warranted in concluding that the ocular lesion is tuberculous, even if a general reaction follows the subcutaneous administration of tuberculin as a diagnostic agent. It is true that the general reaction may furnish a strong presumptive indication, but that is about all we are entitled to say, if the local reaction in the eye is lacking.

DR. OSCAR WILKINSON, Washington, D. C.: I desire to add to Dr. Wilder's remarks that proper food is almost as necessary as fresh air. These diseases occur among a class of patients who are poorly fed. One speaker said he had seen these cases among country folk with plenty of good food. It is well known that country people are not so well fed as city people. They have less variety, and the food is not so well prepared as that of city people. In the second place, we should not forget the importance of intestinal antiseptics. Dr. Bruns taught us that bichlorid of mercury internally was almost a specific. I know that with fresh air, good food, abstinence from sugar and sweets, and with internal antiseptics a chronic case of phlyctenular ophthalmia is an exception.

DR. LEDBETTER: By the time I get through with the diagnosis the patient is well.

DR. DAVIS: Tuberculin does not act as a remedy, it stimulates the body. If you do not get a reaction stop using it. Watch the temperature and watch the patient. You are dealing with a powerful toxin. It is only an adjunct in the treatment.

DR. WILL WALTER, Chicago: The reason for the 10 per cent. failure of positive von Pirquet in these cases, if they are of tuberculous origin—and some take this failure as evidence against such origin—may be explained. These reactions are specific, not only in that homologous antigens must be used, but even identical strains of organisms. In the hemolytic Wassermann reaction several strains of liver extract are now commonly employed and not infrequently we get reports of positive reaction for only two or three of these antigens. Irons has failed to get response in his cutaneous test for gonococcal infection until the same strain was found. He is now using a mixture of several strains. The difficulties in this regard are greater with tuberculin because of changes due to the prolonged time needed for the growth of bacilli *in vitro*. When fatal doses of tuberculin are given to tuberculous guinea-pigs one finds extensive hemorrhages in the foci. I have shown foci in gland and spleen from two guinea-pigs infected three months previous to our test. We gave one a small, and the other a large, diagnostic dose in ratio to body-weight. In the former specimen one sees intense hyperemia and in the latter hemorrhages are apparent. Such reaction of foci in the eye from the subcutaneous test might thus prove perilous to the integrity of vision or even to the eye itself since there is no way of judging, in advance, what degree of reaction may supervene.

TOPICAL DIAGNOSTIC VALUE OF THE HEMIOPIC PUPILLARY REACTION AND THE WILBRAND HEMIANOPTIC PRISM PHENOMENON

WITH A NEW METHOD OF PERFORMING THE LATTER *

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BOSTON

METHOD OF PERFORMING THE HEMIOPIC PUPILLARY REACTION

The usual method of examination of the hemiopic pupillary reaction up to the time of the appearance of the work of Hess was to throw a narrow pencil of light onto the blind and likewise onto the seeing retina, noting which gave the best reaction. Commonly an ophthalmoscope or other small mirror was used to reflect rays into the patient's eye from a light placed back of the patient's head. Ahlström[1] and others used an electric light encapsulated except for a 0.25 mm. diameter opening which gave a narrow cone of light to be thrown into the eye. Friedlander and Kempner[2a] used a lens system to focus the rays at 4 cm. distance from the opening. Similar instruments have appeared in this country.

Wolf[7a] used his electric ophthalmoscope to flash with corrective lenses a sharp image of a section of filament onto a definite part of the retina, at the same time observing the pupillary reaction. Heddaeus made an ingenious but apparently not very popular departure in using two lights of equal intensity exposed alternately without interval at equal distances from the fixation-point and from the eye.

Following the use of these methods, reports more or less favorable to the diagnostic value of a hemiopic pupillary reaction were received, among others from Wilbrand, Wernicke, Schmidt-Rimpler, Seguin, Leydens, Rothman, Henschen, Schwartz, Dercum and Oliver, Salomonsohn, Vossius and Lange; while more or less unfavorable reports are noted from Heddaeus, Brixa, Silex, Liebrecht, Hirschberg, Martins, Bach and Oppenheim, the last three rather giving the reaction the benefit of the doubt than denying its value.

Hess introduced the methods described of hemikinetic illumination of the retina and of concentric illumination about the macula, both of which render the dispersion light reaching the macula as nearly constant as possible.

Sachs,[6a] unaware of Hesse's work, at the same time was using a modification of the same principle. The patient observed the center of a large bright reflecting white mat surface. One half, then the other half, was quickly covered by a black curtain while the pupillary reaction was noted. Since the appearance of these works, reports more or less favorable to the diagnostic value of the hemiopic pupillary reaction have come from Best, Ginsberg and Dessauer, Behr, Krusius and Jess, while those more or less unfavorable have issued from Hess, Hesse, Laqueur and Veraguth.

Since so much depends on the method and apparatus used in examinations of pupillary reactions, it would seem advisable to give in some detail the instruments and method used in examination of the cases which follow. I have been forced to make up apparatus from material readily available about an ordinary laboratory. Certain points may then possibly be of interest to others similarly situated, since the apparatus is not on the market in this country.

After some search and experimenting it was found that certain tin containers to be obtained at any drug-store could be satisfactorily pressed into service.

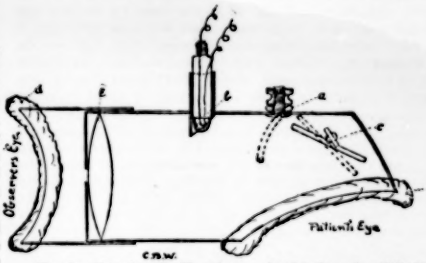

Fig. 1.—Objective Pupillometer: *a*, head-spring; *b*, ophthalmoscope lamp; *c*, mirror; *d*, *d'*, velvet pad; *e*, 14 D. trial-case lens.

Figure 1 represents a considerably modified monocular pupillometer after the principle of Krusius.[8a] It is held in position over the patient's eye by a brass head-spring attached at *a*. A rheostated electric ophthalmoscope lamp (*b*) with frosted globe illuminates the patient's pupil whose image is thrown by the calibrated adjustable laryngoscopic mirror (*c*) into the observer's eye after being magnified by a 14-D trial case lens (*e*). All extraneous light is excluded from both eyes by the

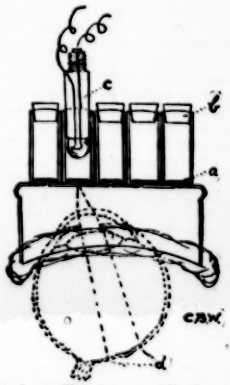

Fig. 2.—Entopic pupillometer: *a*, milk-glass; *b*, stopper; *c*, ophthalmoscope lamp; *d*, dispersion-circle.

thick close-fitting black velvet rim-pads (*d*). By this means an objective examination of either eye can readily be made without interfering with the field of vision of the other eye.

In Figure 2 is shown the more accurate entopic apparatus modified from that used by Hess. A considerable improvement for clinical purposes I have found in the addition of several pin-hole openings which can be

* The major part of this work was performed while acting as assistant on Dr. Harvey Cushing's staff at Johns Hopkins Hospital, Baltimore, during 1911 and 1912. The remainder was done in Boston on Dr. Cushing's neurological surgical service at the Brigham Hospital, Harvard Medical School, and at the Massachusetts Charitable Eye and Ear Infirmary.

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

used at will on either eye to bring the dispersion-circle into the best position for observation, especially when field defects are present. In each case the pin-hole is illuminated through a thin milk-glass or oiled paper (*a*). The resulting dispersion-circle (*d*) falling on the retina is limited in size only by the size of the pupil. The apparatus is fixed over the eye, and outside light excluded in the same manner as that described before. With these modifications many more patients are able to use this method. Only average intelligence is necessary.

Figure 3 shows the form of test-light (*A*) used. The strength of the light may be varied in the first place by choice from a series of lamps ranging from 2.8 volts to 5.5 volts, tungsten filaments, secondly by resistance (*R, R'*) connected in series, and thirdly by regulating the ordinary rheostat current by which the electric ophthalmoscope is operated from the house-current and finally by different sizes of diaphragms (*b*) thereby

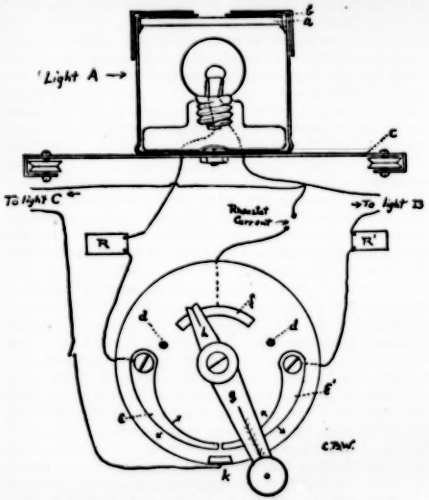

Fig. 3.—Test-light scheme: *a*, milk-glass; *b*, diaphragm; *c*, carriage; *R, R'*, resistance; *e, e'*, movable contact-bar; *d, d'*, stops; *f* and *h*, circuit-breaker; *g*, alternating-switch; *k*, contact-bar for light *C*.

varying the area of illuminated milk-glass (*a*) from any size up to 4 cm. in diameter. This light-capsule is attached to a carrier (*c*) traveling on an ordinary plain perimeter with arms bent back to form a wider arc (from 20 to 30 cm. radius).

For isokinetic, hemikinetic and other tests I have devised a simple switch and wiring system for controlling two exactly similar lights as desired. The switch has two contact bars (*e, e'*) movable as indicated by the arrows so that any interval or no interval, with one contact being made before the other is broken, can be obtained by adjustment. In the position shown in Figure 3 the current will light B. Moving the switch across the interval lights A and turns off B. Either light may be turned off without lighting the other by throwing the switch further over till *h* leaves *f* and strikes either top (*d, d'*) as the case may be. By means of the resistances (*R, R'*) two equal lights can be balanced quickly in an ordinary photometric box, and by adjustment of the switch can be made to show no change of intensity in alternate lighting both in the photometric box and by a further slight adjustment, on the normal pupil by the entopic method. Of course much stronger lights can be used with the same system.

While this method, when the larger diaphragms are used, is satisfactory in performing the hemikinetic test of Hess, it is also desirable to have at hand the instrument of shutter-type as described. I have applied a different style of shutter which has several advantages. The shutter shown in Figure 4 is rotary and not sliding as devised by Hess. This shutter gives a symmetric distribution about the macula, with the weak or peripheral retina receiving the largest part of the image, while the smallest part is nearest the sensitive macula. The shutter works smoothly and accurately and without shaking the instrument as the sliding shutter of Hess may do. Most important of all, the light in changing is cut off and added in increments situated with absolute symmetry with respect to the macula, while with the sliding shutter during the change

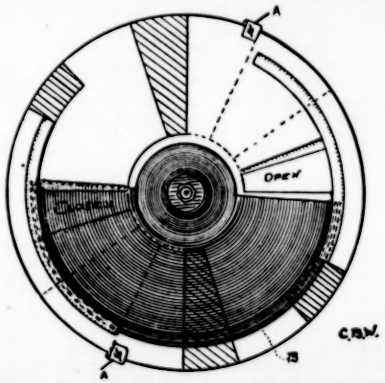

Fig. 4.—Rotary Shutter Disk: *A, A'*, fixing clamps for holding segments in place.

light is taken from one side and added to the other side at a different distance from the macula. This will cause a slight reaction at the time of alternation, aside from light adaptation phenomena. A plain shutter of the rotary type having fixed sizes of light surface is easy to make; but considerable and accurately symmetric variation in the size of the light-surfaces without changing the fixation-point may be obtained by use of segments patterned as shown in Figure 4. These segments cross in the center, scissor fashion, and make up a disk. Superimposition of segments not crossing the center gives a flat surface on which the semicircular shutter may rotate. A fine hole through the central binding-pin serves as a fixation-point. The shutter is operated by a spring lever as in the ordinary rotary camera-shutter.

METHODS OF PERFORMING THE WILBRAND HEMIANOPTIC PRISM TEST

As originally suggested by Wilbrand, neither the strength of the prism nor the distance of the patient from the point of fixation was definitely stipulated. Dif-

ferent observers have varied both the distance and the strength of the prism. Some have used a light as a fixation-point instead of the white spot on a black wall as originally suggested. Heine further suggested a combination of the Wernicke and Wilbrand tests, using as a fixation-point a source of light which was strong enough. To this method it seems to me that there are serious objections which will be considered later.

While I have used the usual prism method, I have used in addition a device which is free from certain difficulties attending the use of prisms. At first a single electric light such as the test light shown in Figure 1 was used. The light-surface was covered with thin black silk gauze or a very thin coat of a diluted black paint so that it was invisible when not lighted on a black background or behind a black screen in a dark or semidark room. It was then regulated to an intensity such that no sense of dispersion-light sheen was notable in the normal eye when the image of the light fell on the blind spot. This light was used as a fixation-point at a distance of 1 or 1.5 mm. usually. Now with a small noiseless switch one may turn off the light in the same instant or the instant before the prism is interposed to throw the image onto the blind retina. In this way one may compare the action of the eye when its blind retina is actually stimulated and when it is not, under the same conditions.

This method was further improved by use of two lights a certain distance apart and the switch shown in Figure 3. In this case a prism may or may not be used, and the distance between the lights may be made to correspond with strong or weak prism values. Finally, by the addition of segments to the switch, three or more lights were used in the same way. Thus the extra segment (*k*) is used to operate the third light (*C*) when the other segments (*e*, *i*) are swung down opposite it. This extra segment is a slightly lower plane than the other segments, and the light (*C*) may be turned out without lighting the others by simply raising the switch handle. By means of an arrangement of this sort one can perfectly control the appearance and disappearance of the fixation-point in any part of the field. Further, in addition to varying equally or unequally the horizontal distance of the secondary fixation-points from the central fixation-point, one can also vary in the same way the distance of the former, above or below the latter in different parts of the field, by attaching the lights to arms movable about the center. This is very important in analyzing the psychologic complex which I believe to be present in this phenomenon.

A detailed report of twelve hemianoptic cases—both anterior and posterior lesions—together with the complete bibliography and history of the subject may be found in the original presentation, but a summary of the examination results in these cases may be given here.

SUMMARY: I. THE HEMIOPIC PUPILLARY REACTION

From a study of the physiologic and physical difficulties in making hemiopic pupillary tests, certain errors become obvious which have doubtless been common to a large number of reports. These have of course been chiefly in the use of single beams of light.

Thus Seguin in his bitemporal cases carried the light slowly from the temporal side to the center. The pupillary reaction was then so gradual as not to be noted until within from 50 to 60 degrees of the center. Now on the nasal or seeing side the light was thrown abruptly over the bridge of the nose, and the reaction noted at once, since it could not be started at 90 but at 60. The obstruction offered by the nose is particularly effective in producing an error in the group of bitemporal hemianopsias in which a hemiopic reaction should theoretically occur. Also the use of the old electric instruments favored variation of the distance of the light-source from the pupil with corresponding pupillary change, there being a tendency again because of the nose to vary the distance on the nasal and temporal sides.

The combination Wernicke and Wilbrand test of Heine is open to several errors. If this light is 20 feet distant and strong enough to give a pupillary reaction it will also give a definite sheen of dispersion-light when thrown on the blind retina so that the patient has a cue which way to move the eye. If it is weaker it must be inside of 20 feet, and when the instant loss of fixation occurs the pupil dilates with disaccommodation and contracts again on refixing, owing to accommodation or convergence, as well as to the light itself. Psychic reactions must of course be carefully excluded.

For clinical purposes in testing for the hemiopic pupillary reaction a rotary hemikinetic shutter of the type described is, I think, the instrument of choice, since the openings are more symmetrically arranged and exposed than the sliding types of Hess, are easily regulated and are absolutely alike as to size, though somewhat more difficult to construct properly. Balanced electric lights with a switch as described are more difficult to keep in adjustment, but their use requires no assistant. The presence of a slight reaction or pupillary jump at the instant of alteration in these methods have been considered light-adaptation phenomena. If present at all, the pupillomotor activity of the peripheral retina is so extremely weak that this explanation would not seem to hold. Indeed, when this phenomenon occurs it is usually caused by faulty mechanism or possibly a psychic or psycho-accommodative effect.

It is notable that failure to elicit hemikinesis has occurred in hypophyseal cases which reached the stage of complete hemianopsia to normal disks only a comparatively short time before examination. That optic atrophy had not progressed far was shown by the rapid recovery after the physiologic or functional block had been removed by operation—in some cases over one-fourth of the temporal field being recovered at the first field examination four days after the operation. Possibly, then, the pupillomotor function in the macular region had not suffered sufficiently to give noticeable hemiopic pupillary reactions. When the macula is artificially blinded so as to reduce the central pupillomotor activity, not only is the fixation likewise greatly impaired, making the examination very difficult if not impossible, but also the patient is apt to blame any subsequent loss of central vision to this procedure.

In case the macular region is damaged we have presumptive evidence, when taken in consideration with the form of the field defect, of an anterior lesion since central scotomas are far more common in these lesions than in posterior lesions. The presence of a central scotoma should in addition to its diagnostic value make the examination of hemiopic pupillary reactions easier, owing to the decreased activity of the pupillomotor macular fibers; but on the other hand the difficulty of fixation makes the examination more difficult. If the central scotoma is in one eye only, fixation may be maintained by use of the other eye with an instrument I have devised,* but it necessitates the

* Walker, Clifford B.: Some New Perimetry Instruments. THE JOUR. A. M. A., July 26, 1913, p. 277.

observation of the direct pupillary reaction. If the results are not consistent they are of course valueless. Indeed, the consistency of the results in Case 1 is the only reason for attaching any importance to them. The possibility in this case that the pupillomotor fibers in the macular region may have suffered in a more or less one-sided manner is interesting, but of course not conclusive from the case cited. It is evident, however, that even if the hemiopic pupillary reaction proves demonstrable in this group of cases its usefulness will be very limited indeed.

Concerning the bearing of the presence of pupillary reaction in central scotoma cases on the theory of Heddaeus and of Hess that only the macular region is pupillomotoric, the findings in Case 7 seem to be particularly illuminating. It is at once suggestive of that group of cases reported as having central scotomas but still retaining pupillomotor activity, although the area of central retina involved was greater than that determined by Hess as the pupillomotor area. In such cases it is easy by the use of two or more disks to determine the size of the scotoma; but to locate the position of the macula in the scotoma is not so easy, and in the case of double central scotoma may be practically impossible. It must be remembered then that the macula may be eccentrically situated in the scotoma so that the entire pupillomotor retina is not damaged. Further, it seems to me that it must be demonstrated that light-perception is nil in the scotoma area although disks cannot be seen to move. To determine the light-perception in a scotoma with a single light is practically impossible since the patient can always detect the light by the sheen of dispersion-light, exactly such as one sees in the dark room when a beam of light is thrown on the blind spot in the normal eye. In determining these points I applied a new method described in the examination in Case 7, using my alternating pupillary lights to get the size of the scotoma for light and then comparing it with the blind spot. It then became apparent that in all probability the area of absolute central scotoma could not involve the entire area of pupillomotoric retina as measured by Hess. Indeed, as a rule it would seem safe to assume that the best test as to whether a central scotoma of this particular size is absolute or not would be the absence of pupillary light reflex, provided there was no other reason to account for the absence other than the scotoma.

II. THE WILBRAND PRISM TEST

I believe that in the Wilbrand prism test the factors of intelligence and habit have not been sufficiently controlled in previous observations. Posterior lesions commonly give homonymous field defects. In the horizontal plane it takes only one coordinate movement of the eyes to bring into view objects on the blind side. That is easily remembered and soon learned by the intelligent patient. It becomes a habit to look on that side for something that has disappeared. Refixation becomes accordingly more rapid than in bitemporal cases which are commonly anterior lesions. In the latter the patient gets little or no training in refixation ordinarily, since the defective temporal field of one eye is aided by the nasal field of the other eye. When one eye is lost or when central scotoma develops in one eye, as is common in anterior lesions, the habit of refixing in the other eye would naturally develop according to the intelligence of the patient.

It would hardly seem fair to compare the refixation ability in these two groups of cases unless the patients have had equal experience and habit-formation. This condition will be fulfilled only by those patients in the bitemporal group who have lost one eye, or the condition might be brought about by blinding one eye artificially until the patient became well accustomed to such a state of affairs. The same would be true of anterior lesions producing hemianopsia in only one eye, but of course would not hold for that rare group of anterior lesions producing homonymous hemianopsia. The latter would be directly compared with the group of posterior lesions.

When these factors are considered and the examination conducted after the method I have described with multiple alternating lights or even with a disappearing object and a prism, several interesting points are noted. In general the refixation-time in all cases increases as the displacement of the object onto the blind side increases, and for each displacement decreases more or less on repetition, provided the patient is intelligent and the displacement is made in the same way each time. If, however, the displacement is varied to different distances above and below the horizontal plane through the fixation object, more or less confusion may be introduced in all cases and a marked tendency may be noted in all cases for the patient to move his eye to the point where the object was last seen rather than directly at the point above or below, as the case may be, where the object may really be refixed. Indeed, this tendency to look toward the previous point of displacement on the blind side is marked even when there is no stimulus whatever, the object in the center having simply been made to disappear. This paradoxical effect of an apparent stimulus arising in the real absence of a stimulus serves well to show the factors of intelligence, observation and habit which may complicate these tests.

While it may be noted that the refixation time is in general greater in the bitemporal hemianopsias of anterior lesions than it is in homonymous posterior lesions, it is also true that patients with the former, if intelligent, reduce their reaction-time more markedly as the number of trials increases. To keep one eye of such a patient bandaged for a length of time and note the effect on the refixation-time in the other eye would be interesting, although I have not yet been able to carry it out, except for comparatively short periods of time.

That the Wilbrand prism phenomenon is a pure reflex receives little or no support in the observation of these cases, while there are several points, as mentioned, indicating that the phenomenon is the functional result of more or less intelligent observation and consequent habit-formation. The form of the field defect has a bearing on these factors as well as on the site of the lesion; indeed, it apparently often accounts entirely for the results of the test, when conducted after the described method. Thus the Wilbrand test would seem to be more of a measure of a certain power of observation than a test for the integrity of a definite reflex arc.

CONCLUSIONS: I. HEMIOPIC CAPILLARY REACTION

1. Nothing has been noted in these cases in disagreement with the theories of Heddaeus and Hess that the peripheral retina lacks pupillomotor sensitiveness.

2. The possibility of a hemiopic pupillary reaction within the pupillomotoric area is suggested.

3. In absolute central scotoma cases the absence of direct pupillary reaction without other cause than the scotoma speaks for the absolute central involvement of an area at least as large as the pupillomotor area, while

the presence of direct pupillary reaction when the central scotoma is greater than the pupillomotor area as tested by large disks requires further tests for light-perception as described.

4. Observations may be complicated by "concentric movement" or other psychic reactions which may sometimes be eliminated by repetition.

5. Clinically the hemiopic pupillary reaction, in those chiasmal lesions of dyspituitary origin, of rather rapid onset and rapid recovery after operation, has failed, possibly due to a certain retention of pupillomotoric function, centrally, after vision for disks has been lost by the hemiopic retina.

II. THE WILBRAND HEMIANOPTIC PRISM PHENOMENON

1. The peculiar distribution of field defects in anterior and posterior lesions often adds to and encourages psychologic factors which greatly complicate the Wilbrand test.

2. The presence of pseudorefixation, as demonstrated by the new method described, throws serious doubts on the presence of a definite reflex arc in the Wilbrand prism-phenomenon.

3. Clinically, the Wilbrand test has offered no valuable diagnostic data in these cases.

All the cases mentioned in the paper were examined in the large neurologic surgical clinic of Dr. Harvey Cushing, to whose kindness and generosity I am deeply indebted for the privilege of presenting this particular phase of these cases. I extend my sincere thanks to Dr. Cushing for stimulating interest in this work, to Dr. George S. Derby, without whose suggestions and encouragement this report would not have been made here, and to Dr. F. H. Verhoeff for friendly discussion and criticism.

ABSTRACT OF DISCUSSION

DR. C. D. WESCOTT, Chicago: Dr. Walker deserves praise for his careful investigation and for the construction of this ingenious apparatus. A careful study of his paper and his conclusions, however, leads me to think that the tests, as developed at present, are not sufficiently accurate to be generally adopted. It seems to me the patient must be of exceptional intelligence to cooperate with sufficient accuracy to make them of practical value.

DR. CLIFFORD B. WALKER, Boston: The fields were taken with this new set of instruments, of which a short description may soon be found in THE JOURNAL.

PREVENTABLE BLINDNESS—A CHALLENGE TO THE PROFESSIONS *

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In a country keen for pleasure and success, the tragic thwarting of a hundred thousand citizens[1] by blindness in a paradox. It is more; it is a challenge. And when we consider that, among these hundred thousand blind, about 25 per cent. are disabled through the failures of three great and old professions—medicine, business and statecraft—and of one upstart profession—social service—then this challenge assumes an ironical air. Do twenty-five thousand persons, by their blindness, deprive the country yearly of perhaps seven or eight million dollars' worth of productive labor, not because of any lack of knowledge and power on our part, but because of our failures in applying the knowledge and power which we possess?

The problem of preventable blindness is in theory simple. It is a problem in the elimination, through the canniest possible organization, of waste: the waste involved in fruitless lawmaking, bungled administration, medical education often unworthy of the name, medical service, here sparse, there prodigal without avail, a myopic industrial system, and social service, hovering on the outskirts, eagerly interested in immediate needs, but dull to the essential need.

As the elimination of such waste in clinics is a point to which I shall more than once recur, let me cite some actual accomplishments. For example, at the Boston Dispensary Eye Clinic, in 1910, one-half of the physician's refraction work was found to be wasted. As Hartshorn and Davis have explained,[3] a trained worker in medical-social service was assigned to this clinic with the following results: In the autumn quarters of 1910 and 1911, the spring quarter and two autumn months of 1912, an average of about 350 patients, per quarter, were found to need glasses. Before the advent of the social worker about half of these patients would have failed to have their prescriptions filled. In the first of these periods, however, only 29.4 per cent. did fail to obtain glasses; and in the succeeding periods this percentage dropped to 20.4, 7.9 and finally 4.5 per cent.

Before the advent of the medical-social worker in 1910, 44 per cent. of the patients with iritis, phlyctenular disease, acute conjunctivitis and blepharitis paid only one visit. In 44 per cent. of these cases, therefore, the physician's work was likely to be lost. Two years later the percentage of lost cases, in the same clinic and with the same medical service, was reduced from 44 to 24.5. In other words, the physician saved about 20 per cent. of wasted effort. As for the effect on the patients, the percentage of those whom the physician considered cured rose from 17, when he worked alone, to 40.5 when he was backed up by a medical-social worker.

In view of these results, is it not safe to say that efficient management in clinics can be made a potent factor for minimizing blindness?

CAUSES OF BLINDNESS AND METHODS FOR PREVENTION

A study[4] made at the request of the Massachusetts Commission for the Blind, in 1910, shows that among 301 cases of practical blindness in three Boston hospitals (1908 and 1909) the causes were as follows:

Diseases.	Cases Per Cent.
Glaucoma	15.3
Injuries	12.0
Uveitis	13.6
Optic atrophy	11.6
Diseases of the cornea (excluding gonorrheal conjunctivitis, interstitial keratitis and trachoma)[5]	10.0
Ophthalmia neonatorum	4.3
Syphilis[6]	6.0
Trachoma	2.3

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

1. About 118,000. This estimate is based on the proportion of blind to the total Massachusetts population found by the Massachusetts Commission for the Blind, whose register indicates 4,275, or 1.27 per thousand population, as against 2,278 recorded by the 1910 census. In other words, it seems to show that the census recorded only 53 per cent. of the actual cases. The fourth annual report of the Delaware Commission for the Blind (p. 16) shows 286 blind as against 138 shown by the 1910 census. In other words, it indicates that the census recorded only 48 per cent. of the actual blind in Delaware.

3. Hartshorn and Davis: Follow-up Work as an Element of Efficient Treatment in a Clinic for Eye Diseases, Boston Med. and Surg. Jour., April 10, 1913.

4. By the Research Department, Boston School for Social Workers, supported by the Russell Sage Foundation.

5. This includes seventeen cases of leukoma, some of them probably due to ophthalmia neonatorum and others to injuries.

6. This percentage is based on the cases diagnosed as syphilitic and is possibly much lower than the facts would warrant.

Assuming, now, as we safely may, that these diseases, in varying proportions,[8] are the main causes of blindness throughout the country, let us consider how best to attack them. Against few, if any, shall we find the purely medical attack quite adequate, while against others we shall find the support of the city, the state and even the nation, a necessity.

Optic Atrophy.—The attack on optic atrophy seems to call for relatively little non-medical support—not that the profession has so far succeeded in greatly reducing blindness from this cause. Optic atrophy, on the contrary, was a sort of medical *bête noire* until lately, when research began to make its etiology somewhat more clear.

For example, in a case which Dr. George S. Derby kindly permits me to quote, the patient would usually be given a careful, but merely local, examination, after which a diagnosis of optic atrophy would be made. As a matter of fact this patient, B. C., was first seen at a Boston hospital last October, since which time he has been under constant ophthalmologic supervision, numerous examinations having been made. The diagnosis was secondary optic atrophy, cause unknown. After several months of examination in medical, surgical, neurologic, laryngologic and ophthalmologic clinics, during which time four roentgenograms were taken, two Wassermann tests were performed and a lumbar puncture was done, a diagnosis of probable tower skull was made by Dr. Harvey Cushing and a decompression performed in the hope of arresting the atrophy.

This case is, of course, unusual. But does not hope of reducing the present percentage of blindness from optic atrophy of different origin depend very largely on just such intensification of diagnostic method? If so, and if accurate diagnoses must often be based on study by varied experts, we are faced by an economic as well as a medical problem. For though B. C., a very poor man, received this expert attention almost gratis, many of us on small salaries, if attacked by optic atrophy, might have to surrender ourselves to hopeless blindness.

If, then, salvation from this disease is not to be restricted mainly to plutocrats and the proletariat, physicians, hospitals, social engineers and philanthropists must unite in providing, throughout the country, available facilities for examination and tests by specialists in the most diverse fields of medicine.

Glaucoma and Trachoma.—While glaucoma and trachoma, from the medical point of view, are obviously poles asunder, as causes of blindness they have one point in common; both seem to vary with race. The Massachusetts investigation appears to have shown that where English and Hebrew immigrants predominate, we may expect to find glaucoma unusually prevalent; while as for trachoma, immigrants from southeastern Europe and from Asia are so disproportionately infected that, other things being equal, this disease will be most prevalent where such immigrants abound. The control of both these diseases must therefore depend partly on control of immigration. In the case of glaucoma, a non-infectious disease, the federal government can be expected to exclude only blind persons or persons threatened with blindness. In the case of trachoma, however, immigrants known to be infected are deported, and, better still, are turned back at the port of embarkation.

Glaucoma, a probably hereditary disease, may some day be limited by eugenic education. Meantime, blindness from glaucoma will result most often in cases for which expert medical care is far to seek. The elimination of blindness from this disease must, therefore, largely depend both on the colonization, so to speak, of ophthalmologists in cities and towns where they are needed, and on a wise distribution of public clinics; and these must be not only accessible, but also almost inescapable. In clinics, new glaucoma patients may, for example, be "calendared" at the social worker's desk, so that they will be automatically followed up if they fail to return for treatment on the day which has been set by the physician. By the employment of this simple method delays of which the results would be tragic can easily be prevented.

Injuries.—The prevention of injuries, which account for some 14 per cent. of our cases of blindness, has already been shown to depend in part on such complex industrial factors as workmen's compensation and factory inspection laws, public education, active safety work by employers and employees and facilities for first aid and expert medical treatment. Outside of the realm of industry, the prevention of holiday injuries and injuries at home, in play, etc., something may be done both by law and by educational work in schools and through clubs and associations. Against disablement by both the industrial and the non-industrial type of injury, vocational guidance may also be made a potent weapon; for the Massachusetts study shows that these injuries, superposed on cases of disease, do from two to three times as much damage, in terms of binocular disablement, as is done by injuries occurring singly. In other words, men who are practically one-eyed already, form more than half of those whom injuries reduce to practical blindness. For this reason, if for no other, it is highly important, first, to prevent monocular blindness, whether from accident or disease, and, second to keep one-eyed men and boys, whenever possible, out of occupations dangerous to eyesight.

Gonorrheal Conjunctivitis and Ophthalmia Neonatorum.—A perceptible cause of monocular blindness in adults, gonorrheal conjunctivitis, with the less common pneumococcus and other infections, is far more serious among infants. The usual estimate of 10 per cent. of all cases of blindness is perhaps not far from the facts in most parts of the country. It was shown last year, however, that blindness from ophthalmia neonatorum can be reduced as low as 2 per cent. of all the new cases of blindness occurring in a state of three million inhabitants.[9] Instruction and supervision of obstetricians, public-health education, the required reporting of symptoms by physicians as well as midwives and public supervision of the treatment of reported cases—these and other well-known measures should gradually make ophthalmia neonatorum as rare as smallpox, as a cause of binocular blindness, and very unusual even as a cause of monocular blindness.

Syphilis.—The Massachusetts study seems to have shown that syphilis, both acquired and congenital, is responsible for a higher percentage of practically blind patients than one might perhaps infer from the accepted figures for total blindness. Now if syphilis is responsible for 6 per cent., or even half that proportion, of the

8. Stricker, Louis: Blindness and Conservation of Vision: a Sociologic Study, the Civic News, Cincinnati, March, 1913.

9. This statement is based not only on reports to the Massachusetts Commission for the Blind and data furnished by the State Board of Health, but also on 144 answers to a special letter of inquiry sent to hospitals and oculists and general physicians throughout the state.

practically blind in a typical large city, it is not enough to make a diagnosis, or even to inform the average patient that constitutional treatment is necessary. We must somehow make certain that the disease at the root of the local symptoms is actually and thoroughly treated.

Especially in cases of interstitial keratitis, as Derby and Walker[10] have recently pointed out, eye disablement, as well as other serious disabilities, can often be prevented only by the most persistent of social follow-up work. Without this work, such cases as the following are frequent: A little girl of 8 appeared at the Massachusetts Charitable Eye and Ear Infirmary with interstitial keratitis. The treatment was local. Then followed treatment in twenty-eight different clinics all over Boston—nose, throat, lung, children's and surgical—as various symptoms appeared. Treated singly and without reference to the underlying cause, the symptoms of congenital syphilis made this child a "clinic trotter" along a zigzag path leading anywhere but to health. But when taken in charge by ophthalmologist syphilologist and social worker together, this child received persistent constitutional as well as local treatment. Her eyes, ears and general condition all markedly improved; and the child, as it now appears, was saved from partial if not total blindness.

Uveitis.—All cases diagnosed as syphilitic or tuberculous having been separately classified uveitis in our Massachusetts study, usually refer us back to cases of iritis, iridocyclitis, chorioiditis, etc., of uncertain etiology. If uveitis, as our results seem to show, causes about 14 per cent. of the cases of practical blindness, further measures of prevention are essential. Now few, if any, of the patients in question would have lost all useful vision if they had received early, intelligent and thorough treatment. Once more, therefore, our problem seems to be very largely social and financial—the problem of placing ophthalmologists where they are most needed and of making clinics more effectual by the elimination of medical waste.

Diseases of the Cornea.—Excluding gonorrheal conjunctivitis and ophthalmia neonatorum, interstitial keratitis and trachoma, we find that diseases of the cornea, mainly keratitis of uncertain etiology, and corneal ulcer, often of traumatic origin, are apparently chargeable with 10 per cent. of our cases of practical blindness. Results of this kind at the hospitals from which our data were gathered must mean that patients either neglect or waste the medical service provided. If similar results prevail in other parts of the country, it is again obvious that medical facilities must not only be made accessible when needed, but also so managed as to prevent these diseases from contributing largely to the total of practical blindness.

We have now glanced at the chief causes of preventable blindness. In so doing we have seen that the preservation of eyesight from each and all of these causes involves not only medical skill, but also education, law and its administration, social service work, and a wise distribution of medical facilities. If we went on to consider remediable causes of blindness, such as senile cataract or congenital cataract, or such secondary causes as measles and meningitis, we should find the same conclusions true. If brief, then, it is safe to say that, in the whole field of work for good eyesight, social organization, in the broadest sense of the term, must supplement and support even the best of medical efforts.

10. Derby, George S., and Walker, Clifford B.: Interstitial Keratitis of Luetic Origin, Tr. Am. Ophthal. Soc., 1913.

STEPS WHICH OUGHT TO BE TAKEN

What plans of work and what forms of organization should now be adopted?

There are organizations for the conservation of eyesight in Arkansas, California, Indiana, Kentucky, Maryland, Massachusetts, New York, New Jersey, Ohio, Pennsylvania and Wisconsin. Through answers to direct inquiry, we learn, however, that their work has so far been mainly confined to public education through pamphlets, lectures, etc., except in Kentucky, Massachusetts, New York, Ohio and Wisconsin. Perhaps the most distinctive achievements in these states have been:

1. In Kentucky: The "Mountain Fund," so called, and the traveling clinic by which Dr. Stucky and his assistants have carried into the mountains their campaign against trachoma and other eye diseases and defects.

2. In Massachusetts: (*a*) Studies of causes of blindness and partial eye disablement; (*b*) the joint campaign by which the State Board of Health and the Commission for the Blind have secured a relatively strict enforcement of the ophthalmia neonatorum reporting law, the following up of all reported cases and legal or other action against persons involved in cases of partial or total blindness due to ophthalmia neonatorum and neglect; (*c*) the consequent reduction of ophthalmia neonatorum, as a cause of blindness, to 2 per cent. of all new cases; (*d*) extension work by the Commission for the Blind through its field agents in teaching patients, charitable agencies, etc., in the remoter districts, how to make the most of existing facilities for treatment; (*e*) the establishment of an evening pay eye clinic in Boston; (*f*) the gathering of information and the reduction of waste by the instalment of social service departments in eye clinics; (*g*) the opening of a special class for children with defective eyesight by the Boston School Board, and (*h*) the utilization of the workmen's compensation law as a practical measure for the conservation of eyesight.

3. In New York: (*a*) improved training of midwives; (*b*) special public schools for children with trachoma; (*c*) special teaching in the regular public schools for children with defective vision; (*d*) a recent investigation of wood-alcohol poisoning; (*e*) factory-illumination legislation and legislation for the prevention of accidents, and (*f*) not only a modified system of reporting for syphilis and gonorrhea, but also an appropriation of $55,000 in New York City for a hospital for the treatment of these diseases.

4. In Ohio: (*a*) the campaign of the State Commission for the Blind against ophthalmia neonatorum, including the employment of nurses to follow up cases, and the prosecution of delinquent midwives.

5. In Wisconsin: (*a*) the required use of a preventive at every birth, (*b*) the follow-up work for cases of ophthalmia neonatorum begun by the Department of Child Hygiene of the Milwaukee Board of Health, and (*c*) activity by the Industrial Commission promoting safety work among both employers and employees.

From this summary it certainly seems probable that these are the states so far contributing most to our campaign. And, if so, is not the question of organization very nearly settled by experience? For in these states, and in these states only, are there state-wide organizations with paid executives. The experience of these

states seems indeed to amount almost to proof that work for the conservation of eyesight can best be carried on, not merely by committees, whether exclusively medical or partly lay, but also through the services of executive agents.

Such agents seem to work most ingeniously when stimulated by association with persons blind, becoming blind or recovering from blindness. They will therefore generally be associated with schools, or directed by organizations, for the blind. And as effectiveness requires coordination of the work of varied state and city departments, whether of health, labor, education or law, these agents can probably best work under state commissions for the blind, such as that created by the Ohio statute. The practical problems of conserving eyesight, however, are so complex and varied that their developments must be supervised by a committee of experts—ophthalmologists and other medical specialists, public health experts and experts in education, industrial safety work, etc. This type of organization is perhaps the most efficient to date.

Whatever the ideal may be, each state must freely work out its own form and its own methods of internal organization; but in doing so each state should have the opportunity of testing its plans and its achievements by matching them up with the plans and achievements of other states. For busy men and women such comparisons are at present difficult almost to the point of impossibility. To make such comparisons easier, some form of interstate organization is clearly needed.

CONCLUSIONS

The facts which I have cited seem to indicate this conclusion: The problem of unnecessary blindness, which has so largely baffled and sometimes even disgraced the professions of medicine, business, statecraft and social service, demands for its solution the following measures:

1. A campaign of medical and lay research and education, government action, medical treatment and social work carried on, wherever possible, to limit and to remedy ocular disease, especially the more prevalent and damaging diseases and defects.

2. In the work of public education, special stress to be laid on the importance not only of ophthalmia neonatorum and trachoma, as causes of blindness, but also of glaucoma, syphilis and ocular injuries.

3. Medical schools to emphasize the interrelation of ocular and general disease, to teach their students the importance of consultation with ophthalmologists and a high sense not only of human service but also of the physician's duty to uphold the health laws, both national and state.

Hospitals and clinics urged to provide ophthalmologists with every possible facility for the diagnosis and treatment of obscure cases, including the help of pathologists, syphilologists, brain specialists, Roentgen-ray specialists, and others.

4. The prevalence of blindness studied in each state in relation to its more prevalent causes, on a basis of reports of cases of blindness to state commissions or other central agencies.

5. To promote such intelligent study of the prevalence and results of eye diseases, a committee of the American Medical Association to request hospitals and ophthalmologists to include in all their eye records the following data: age; sex; single, married, widowed or divorced; birthplace; birthplace of parents; occupation or school grade; in possibly syphilitic cases, the number of children, still-born, dead or living, and in traumatic or possibly traumatic cases, the tool or machine used, and the manner of injury.

6. To facilitate the comparison of results the following standards of vision officially adopted: for blindness, the German standard, vision fingers 1 foot or less; practical blindness, vision less than 20/200 with glasses; partial disablement, vision 20/200 to 20/50, inclusive, with glasses.

7. Making it the duty of the state boards of health or of special private agents to study the needs of all localities; and in cooperation with medical schools and hospitals, to bring to the notice of young ophthalmologists opportunities for work in places especially requiring their services.

In remote districts, where peculiar conditions require it, such clinical expeditions as those of Dr. Stucky in the Kentucky mountains.

In large cities, self-supporting evening clinics, with paid ophthalmologists in attendance, organized to compete with charlatans and inefficient practitioners and to provide self-respecting patients on low wages with facilities for expert treatment otherwise hard to secure.

In clinics and hospitals, the specialist's work made more efficient by insisting on proper treatment of diseases underlying local eye symptoms and by keeping patients under treatment long enough to get results.

8. Paid social workers made part of the regular staff of all eye clinics so as to increase their efficiency in the prevention of blindness from glaucoma, uveitis, etc., and to economize the work of the physicians in charge, as fast as the necessary funds can be secured.

9. In schools, the pupils' eyes regularly examined, preferably by trained physicians; records kept showing the effect of the school curriculum on eyesight, and parents induced or compelled to furnish necessary eyeglasses or treatment for the eyes. If necessary, the state "neglect law" amended to make this possible. In the management of institutions, state and local authorities to set a standard of work for the preservation of eyesight.

Especially in reform schools and prisons, in which the inmates are shut away from the usual medical facilities, ophthalmologists regularly in attendance; and close cooperation secured between ophthalmologists, general physician and the probation officers having oversight over discharged prisoners. All male gonorrheal, and all syphilitic patients detained until cured.[40]

10. Classes, especially adapted for children with defective eyesight, organized in all large cities, not only to provide education for children not properly inmates of institutions for the blind, but also to aid in the preservation of their sight.

School children with the vision of both eyes defective, or with one eye practically blind, guided toward occupations relatively free from ocular injuries.

11. State birth registration laws amended to secure birth notification within three days, and preferably within forty-eight hours; and warnings as to the danger of ophthalmia neonatorum sent to all mothers immediately on receipt of birth notices.

Obstetric clinics promoted to furnish adequate nursing and medical service to the poor in large cities. Midwives trained, registered and supervised in cities in which their services are necessary.

A suitable prophylactic distributed free to all physicians and to midwives. The use of this prophylactic required at all births in lying-in hospitals or in the

40. Massachusetts Revised Laws, Chapter 75, Section 48.

practice of midwives, and at least recommended strongly to physicians.

The symptoms of ophthalmia neonatorum defined by the American Medical Association as redness and swelling of the eyelids and unnatural discharge from the eyes, within thirty days of birth, and made reportable by physicians as well as nurses and parents.

Physicians, as well as midwives, prosecuted whenever necessary to secure observance of the reporting law; and physicians admonished by their state societies for failure to obey the law or to secure adequate treatment and nursing for serious cases.

Local boards of health given ample powers over reported cases, subject to the supervision and control of the state boards in requiring adequate standards of treatment. Minimum requirements defined by the American Medical Association as the attendance of an ophthalmologist, or of a physician experienced in the treatment of ophthalmia neonatorum, assisted in all but the lightest cases by at least one trained nurse.

12. Trachoma made reportable in all states in which it is prevalent, proper treatment insured under public supervision, and in case children are affected, special schooling provided.

13. Workers for conservation of vision to join with associations for sex education, etc., (*a*) in setting adequate standards of treatment for syphilis, congenital and acquired; (*b*) in raising the physical standards of marriage, and (*c*) in urging the federal government to extend to syphilitic patients the system of exclusion *at the port of embarkation*, now applied to emigrants with trachoma.

14. Legislation against holiday injuries, etc., and popular education as to the dangers of non-industrial injuries of the eye.

Cooperation with both employers and employees in the elimination of industrial injuries.

Team-work with the American Association for Labor Legislation, to secure the enactment or amendment of factory inspection and workmen's compensation laws, so as to provide for study of industrial conditions affecting eyesight, protection from injury and disease, and special compensation, not merely for "total and irrecoverable blindness" of either or both eyes, but also for practical blindness, defined as reduction of vision to one-tenth of normal with glasses.

15. A joint committee of the American Medical Association and the existing state organizations for the prevention of blindness and the conservation of eyesight to provide for interchange of information through periodic reports from each state, published in ophthalmologic journals and the *Outlook for the Blind*, and as soon as any vital demand is manifest, to promote a federation of all state committees and commissions actively at work for the preservation of eyesight.

ABSTRACT OF DISCUSSION

DR. W. H. WILDER, Chicago: Two or three points in Mr. Greene's excellent address, I think, might bear reiteration to some who dwell where this work has not been done with such thoroughness as in Massachusetts. One of these points is the great importance of educating not only the members of the community but also the members of the profession. This is, after all, a campaign of education, and surely in some localities the medical profession is greatly in need of education along this line. In the past there has been altogether too much indifference shown to the recommendations and suggestions of specialists in the medical profession and it has been only by the representations of certain members that the public has been awakened to its importance. We now see that these efforts need further application and emphasis. The New York statute requiring that ophthalmia neonatorum be reported to a physician is the result of Dr. Howe's efforts. I think possibly eleven states now have a similar statute. We have such a statute in Illinois, but it needs enforcement, and furthermore, it needs some changes so that the board to whom it is entrusted will have more police power. The law in Illinois makes it incumbent on midwives to report sore eyes in a new-born child to the nearest physician. The inefficacy of such a law is shown, for often the physician does not know much more about the subject than the midwife. This excellent presentation illustrates the great importance of having some person or persons authorized as a board of commission to carry on this work. Such a commission should be legalized and it must have certain plenary powers which are not ordinarily granted to associations which are outside the government. In Illinois we have been working for some time to bring about, if possible, the establishment of an association for the conservation of sight and the prevention of blindness. We are trying to secure a commission for the blind and a recommendation was made to the state legislature a few years ago, but other questions, backed up with lobbies, took up more time and the medical laws were pigeonholed. It seems to me we must have a commission like that of Massachusetts or Ohio, whose duty it will be to see that births are promptly registered and a census of the blind taken.

DR. CASEY A. WOOD, Chicago: The Council on Health and Public Instruction of the American Medical Association has taken up this important matter and has done the very things that Mr. Greene refers to. An endeavor will now be made to bring into use the machinery of the American Medical Association as a whole in correlation with the state societies. The committee appointed by the council will make use of the experience of such men as Mr. Greene and his commission in Massachusetts and apply the experience gained there to other states and I think much will be accomplished. This movement will be backed by a certain amount of money if the American Medical Association can be shown that actual work is being done. Incidentally it will help to relieve the country of a disgrace—the lack of vital statistics.

DR. THOMAS A. WOODRUFF, Chicago: If Mr. Greene will look up the law passed by Congress I think he will find that any employee injured in any factory has to be taken care of by that factory. The factory cannot settle with him. He may settle with the factory for $200, but the factory will have to take care of him the rest of his life if he is disabled and if he knows enough to obtain his rights. In regard to the education of the profession and the people, I think it is much more important that the common people, before being educated in this, receive education along the line of what is necessary to prevent blindness. That will soon educate the general practitioner in looking after them. In Illinois we recently formed an association for the prevention of blindness. Not only physicians, but also the laity have been asked to join, and a large preliminary organization has been formed. We have the Association of Commerce of Chicago interested in the education of the public and next fall shall have one evening devoted to the prevention of blindness. We have also enlisted the interest of the Chicago *Tribune*, which will, in the autumn, devote space to the consideration of this matter. We have enlisted the *Daily News*, which is giving a course of lectures in the public school buildings of the city devoted to various subjects. Some of these lectures will be devoted to the conservation of vision and the prevention of blindness and we are trying to make it a live subject.

DR. LUCIEN HOWE, Buffalo, N. Y.: A recent investigation has shown that a little over 50 per cent. of the cases of blindness are due to the two venereal diseases. It strikes me that we should join in the prevention of syphilis and gonorrhea and stop that stream at its source instead of trying to dam it back after it has gained headway. We should not mince matters at all in speaking to the public, but should have them understand what the principal causes of blindness really are.

MR. HENRY COPLEY GREENE, Boston: I wish to disclaim any intention of setting up Massachusetts as a model. I particularly hope that physicians organizing in other states will not copy our law, which is defective. The Ohio law is much better. It provides for preventive work definitely, instead of in merely general terms.

APPARENT ESOPHORIA AND ITS RELATION TO CONVERGENCE INSUFFICIENCY

H. B. LEMERE, M.D.
OMAHA

A class of cases has been observed in which there is an esophoria for distance combined with a marked insufficiency of convergence for near. In such cases, it has seemed to my colleague, Dr. J. M. Banister, and myself, that the paradox is entirely artificial, and we have considered the insufficiency for near as the essential state, and the seeming esophoria for distance as fictitious. To such instances of esophoria we have applied the term "apparent esophoria."

I at first used the term "pseudoesophoria" for this condition, but Dr. Savage called my attention to the fact that he had already used this term in referring to a different condition than that which I have described. To avoid confusion I have changed the term "pseudoesophoria," which I at first used in the classification of these cases, to that of "apparent esophoria."

Quoting Dr. Savage:[1] "When the far test shows orthophoria and the near test shows exophoria the error is pseudo in character. . . . The same explanation applies when there is esophoria in the far and exophoria for near." He therefore calls the condition we are considering a pseudoexophoria, and evidently considers the esophoria in the distance as the key to the situation and the source of trouble, and the convergence insufficiency called by him pseudoexophoria as of secondary importance. We believe exactly the reverse: that the convergence insufficiency is the true cause of the symptoms complained of.

It has been repeatedly noted in our practice that in this apparently paradoxical state, when the convergence insufficiency for near has been relieved, the esophoria disappears. The existence of this apparent esophoria should with reason be looked on as spasmodic and due to the increased effort required to secure a sufficient degree of convergence for the near point to avoid crossed diplopia. This effort cannot be sufficiently relaxed when viewing the test object at 6 meters, and the spasm remaining causes a tendency to crossing of the visual lines at a point short of the test object.

We have been so impressed with the truth of this explanation in these cases that we do not hesitate to correct the convergence insufficiency by operative treatment on the internal rectus or by prism exercises in the less serious cases. The treatment is carried out without regard for the apparent esophoria for 6 meters. As a case in point the following history is briefly quoted:

Mrs. H. C., aged 28 years, suffering from severe asthenopic symptoms, manifested under cycloplegia the following refractive condition:

R. + 25 D. S. ⌒ + 25 D. cyl. axis 105.
L. + 25 D. S. ⌒ + 25 D. cyl. axis 90.
Distance Esophoria with full accommodation 6°.
Distance Esophoria under cycloplegia 4°.
Convergence insufficiency—crossed diplopia at 18 inches.

Ignoring the apparent esophoria, we shortened the internal rectus muscle of one eye by the tuck method with the result that perfect balance for distance was secured together with sufficient amplitude of convergence for comfortable work at the near point. The small degree of refractive error was corrected.

Much confusion may result from the difference in the type of the findings in our muscle tests for distance and near if this point is not borne in mind, namely, that the convergence insufficiency gives the key to the situation and that in attempts to secure relief our efforts should be in this direction, leaving the pseudoesophoria out of the scope of treatment.

We are convinced that this confusion in diagnosis and the resulting uncertainty of treatment come, in a large measure, from placing too much value on the stability of the muscle tests for distance, and therefore not giving sufficient consideration to the degree of the amplitude of convergence.

If we find an apparent esophoria when testing at 20 feet, and at the same time uncover a convergence insufficiency for the near point, we may as a rule suspect the condition of the esophoria to be spastic, and adapt our treatment accordingly.

In testing the distance balance we use the Maddox rod with the candle at 6 meters, finding the amount of unbalance by means of prisms. We also use the Stevens phorometer both by itself and in conjunction with the Maddox rod. In testing the near balance we use the Graefe dot and line, measuring the balance by means of prisms.

We have found, however, that these tests often give quite variable results; even more unreliable are the results of attempts to obtain the convergence at 6 meters by means of increasing strength of prisms till diplopia is reached. Banister in testing 100 healthy men found an average adduction for distance (6 meters) of 14.1. Yet if repeated examinations had been made in the same cases the convergence would have undoubtedly been trained so that the average would have shown a constant increase.

We make our diagnosis, therefore, from the results of the measurement of the amplitude of convergence, finding the punctum proximum by noting the near point of binocular vision for a dot, and estimating the punctum remotum by the power of divergence for 6 meters (Nagel).

The question will be raised whether in increasing the efficiency of the convergence we may not also increase the distance esophoria to such a degree that asthenopia or diplopia may result for distant vision; but this fear we have found to be groundless, the distance esophoria often disappearing as the convergence power for near is increased.

In our operative work for the increase of the dynamic power of convergence we have always operated to increase the power of the internal rectus and never to lessen the power of its opponent. We use the tuck operation of Savage with the catgut suture as recommended by Valk, with this modification which, I think, is original with us and which we have found very valuable.

After taking our tuck we do not tie the final knot till we examine the muscular balance with the light at 6 meters. If an overeffect is produced, we loosen and take a smaller tuck and test again. We have found, however, that the main difficulty with the tuck operation lay in those cases in which a very marked effect was desired. Here the deepest possible insertion of the

suture, taking up the greatest possible length of muscle, is sometimes inadequate. If after testing we find this to be the case, we make an additional supplementary attachment with our original suture, which still has the needle on it. This can readily be done at this stage, as the muscle is now pulled forward by the first reefing of the suture. We have taken as many as two such reinforcements of our original suture, and consider this modification indispensable for this work. There has never been the slightest trouble in regard to the increased amount of catgut to be absorbed, and never has any hump or lump ultimately remained at the site of operation.

There are certain cases of convergence insufficiency in which useful working vision may be obtained only by means of operation. If one of these patients happens to have at the same time an esophoria for distance, this esophoria should not deter us from giving this patient relief by taking a tuck in the internal rectus.

I wish to acknowledge the kind assistance of my partner Dr. J. M. Banister. Many of our cases were worked out together and the ideas in this paper resulted from our frequent discussion of these cases.

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ABSTRACT OF DISCUSSION

DR. G. C. SAVAGE, Nashville, Tenn.: I think Dr. Lemere has done well in changing his expression "pseudo-esophoria" to "apparent esophoria." In my books I have used the expressions "pseudo-esophoria" and "pseudo-exophoria" as descriptive of conditions dependent solely on the relationship between the center of accommodation and the center of convergence, the former always existing in association with hyperopia, either increasing an intrinsic esophoria or showing an esophoria in both far and near when there is orthophoria, or showing itself in lessening an intrinsic exophoria. The glasses correcting hyperopia invariably cure this form of pseudo-esophoria. There is still another kind of pseudo-esophoria dependent on weak ciliary muscles, that is, ciliary muscles that have not been well developed. Exercise of the weak ciliaries will invariably cure this form of pseudo-esophoria. Of course this form shows itself in the near in excess of any esophoria that may exist in the distance, or in cases of orthophoria for distance, or even of slight exophoria for distance. Pseudo-exophoria, when dependent on myopia of high degree, shows itself only in the near and the same is true of that other form of pseudo-exophoria dependent on hyperdeveloped ciliary muscles. Pseudo-exophoria, whether dependent on one or the other of the causes mentioned, is never very great in quantity, that is, not more than six degrees, usually less. Higher degrees of exophoria in the near, when there is esophoria or orthophoria in the far, or a slighter degree of exophoria in the far, cannot properly be classed as pseudo-error. I have been led to think, and have so taught in "Ophthalmic Myology," that such cases are dependent on a subnormal development of the convergence center. I have seen cases, and doubtless some of you also have, in which there was no convergence center. The fourth conjugate center would make both eyes sweep to the right and the fifth conjugate center would make the eyes sweep to the left, showing that the interni were not at fault, when there was complete absence of convergence power. If the convergence center can be absent and if it can be hyperdeveloped, then this center, which is the third conjugate center, can be subnormally developed. This would necessitate a great amount of work of such a center in converging to procure binocular single vision in the near. If there are more than six degrees of exophoria in the near then a part of this exophoria is intrinsic and would show itself in the distance if there were no spasm of the interni to counteract it. The fact that Dr. Lemere has found shortening the interni curative in cases of esophoria in the far with exophoria in the near is proof, to my mind, that the esophoria was spasmodic. If it had not been spasmodic, he would have increased the esophoria in the far while lessening the exophoria in the near. I have gone over the last 200 cases of refraction on my record books, and have found some unexpected things. There were 4 cases of esophoria in the far with exophoria in the near, 50 cases of orthophoria in the far and exophoria in the near, 10 cases of exophoria in the far and greater exophoria in the near, 17 cases of esophoria in both far and near, 41 cases of hyperphoria existing alone or complicating lateral orthophoria or some lateral error, 68 cases of orthophoria, far and near, 1 case of paralysis of one or more lateral muscles and 9 cases of esotropia; there were no cases of exotropia. In the 200 cases there were 9 cases of blindness in one eye

It is not against Dr. Lemere's theory that I have found orthophoria in the far and exophoria in the near, for in these cases there may have been an intrinsic exophoria corrected by a spasm of the interni, nor is it against his theory that some of my cases had exophoria in the far with more exophoria in the near, for the lessened exophoria in the far may have been due to a spasm of the interni. My treatment of the cases for want of convergence has not been operative but by means of exercise only. The exercise which I have resorted to has been fully set forth in "Ophthalmic Myology" as "the candle exercise." The patient lights a Christmas-tree candle, and holding it at arm's length, fixes both eyes on the blaze, continuing this fixation as the candle is brought within 7 inches of the eyes. Then the eyes are closed, the candle moved to one side and the gaze immediately thrown into the distance. I begin with three movements of the candle the first night before going to bed and add one every night until fifteen movements have been reached, and after continuing the fifteen movements at one exercise, for one month, just before going to bed, I direct the patient to also move the candle ten times toward himself every morning. This exercise, when faithfully followed, in most cases finally brings a cure which doubtless comes from a development of the cells constituting the center of convergence, also developing the interni so as to make them respond more readily to a slighter stimulus. If the operation of muscle-shortening will do the work, then the patient is saved much time and annoyance. The suggestion growing out of Dr. Lemere's experience is worth considering. I am proud of the shortening operation described in his paper, because forty of my colleagues have claimed it, the first one to make the claim being Dr. Valk of New York. I quote the following from a letter received from Dr. Valk written Aug. 15, 1895. "Dear Doctor.—Your letter is just received. I looked up the editorial you referred to in the *Record*, March, 1893, and now have no doubt that you are entitled to the credit of what seems to be an excellent operation. . . . I will gladly do all I can to correct the matter, and should I refer to it again will always give you credit for it." The other claimants, as a rule, have not made such clear and clean acknowledgements. To the fortieth man, I wrote a few days ago: "You are the fortieth to claim the operation of muscle-shortening. I do not know what the other thirty-nine will think about your claim, but yours is as strong as theirs, yet none of you have the slightest claim to the operation, for it is my own."

DR. S. D. RISLEY, Philadelphia: Many years ago I had the audacity to write that we should probably never discover a law for ocular imbalance for the following reason: If we accept two emmetropic eyes, with normal acuity of vision in each, with a physiologic range of accommodation and convergence as the ideal condition for comfortable vision, then any departure from this ideal condition must be regarded as a defect or an abnormality. Examination of a large number of hatters' forms will show great variety in the form of the human skull and the rarity of a model form. The examination of a large number of dried skulls showed that there was a definite relationship between these abnormal varieties in the outline of the skull and the shape of the orbits and the bony sinuses surrounding the orbital cavities. It is reasonable to suppose that, during development in the anomalous-shaped

V. 191

orbit, certain modifications would occur in the form of the eyeballs which would change their dimensions and consequently alter their radii of curvature, and would also lead to changes in the length and line of direction of the optic nerve and the origin and attachment or length and direction of the ocular muscles—conditions, one or all of which, would lead to more or less marked disturbance of motility and in many complex ways modify the condition of binocular vision. Since there is an infinite variety in the possible deformities in the bony skull so an infinite variety of abnormalties of binocular balance is possible, which, so far as I see, can have no relationship to fusion centers based on undemonstrable hypotheses. We can, however, from a study of well-known physiologic factors, separate the cases of binocular imbalance into two groups, which I designate as relative and absolute insufficiencies. These relative insufficiencies have been accounted for in different ways in this discussion, and are, I believe, often misunderstood, but were recognized and correctly explained by Donders in his great treatise. For example, a child is born with hypermetropia, with or without astigmatism. Experience has taught him to disassociate the convergence and accommodation, that is, he is required to use a part of his accommodation even for distant vision where convergence is not required. If the hypermetropia, for example, equaled 2 D., he must use 2 of accommodation and no convergence, while reading at ⅓ of a meter he must use 5 of accommodation and only 3 of convergence. When his hypermetropia is corrected by a +2 D., the habit of a life-time is disturbed and he is compelled to learn a new trick of vision. He must either relax his accommodation to secure clear vision at a distance, and see double, or secure single vision at the expense of a blurred image in each eye. If the child sees clearly he has a relative insufficiency of the interni. If he secures single vision, he has a blurred image and usually a relative esophoria. These are the cases which may be corrected with prisms by well-directed practice. The absolute insufficiencies, however, belong to an entirely different group and are dependent on faulty attachments of the muscles to the globe itself and no amount of exercise with prisms or otherwise is of permanent value.

DR. EDWARD JACKSON, Denver: If we take brain centers in the physiologic sense, somewhat similar to that in which the terms x and y are used in algebra, they offer a convenient way of working out such problems. They simply mean that impulses converge from all parts of the nervous system to do certain things, and what has been observed in pathology, interference with function by destruction of a certain part of the nervous system. Thus understood the terms Dr. Savage has brought into the discussion explain better than any others I could frame the class of cases to which Dr. Lemere has called attention. An overworked portion of the nervous system, inadequate to the work of convergence that is required of it, will explain a tendency to spasm. I do not know how many members of the section have weeded gardens, but when I was about 6 years old it is said I objected to it because I had to "slant my back" so much. A strained, difficult position kept up for some time requires increased time and effort to resume the normal attitude. I think these cases of esophoria at distance with insufficient convergence belong in that category. The paper does not state the patients' occupation or how the eyes were used for near work. But certainly a large number of patients overwork the convergence in their habitual occupations and I take it that many of this series are cases of inability to relax the convergence because it is weakened by overwork.

DR. CASEY A. WOOD, Chicago: Accommodative spasm is often a matter of overworking a tired-out muscle system. When convergence and accommodative power are not well balanced, and fatigue of one or the other is present, an overinnervation occurs in an attempt to overcome the difficulty. But this, far from improving the situation, only adds to the patient's discomfort and we have the symptoms of accommodative (and perhaps) convergence asthenopia.

DR. OSCAR WILKINSON, Washington, D. C.: Apparently there is a discrepancy in Dr. Lemere's statement. He first says: "The treatment is carried out without regard to the apparent esophoria for 6 meters." A little further on he says: "After taking our tuck we do not tie the final knot until after we examine the muscular balance with the light at 6 meters." If he takes no notice of the esophoria before the operation why should he take note of it during the operation? Why should he not make the test near if he wishes to test the amount of correction of the muscle? I agree with Dr. Risley in regard to the effort to relieve this class of cases by milder means. Of course, once in a great while we are compelled to operate, but in the matter of exercises, as pointed out by Dr. Savage, I use a black dot on a white paper and I get results, notwithstanding Dr. Savage's claim that it is not so effective as the candle, and I use it more frequently. The only question is in regard to the frequency of use. If you overexercise, the muscle becomes fatigued and its strength is not increased. One thing should be borne in mind: When you have an exophoria for near you cannot use as strong plus lenses on that patient as in a normal case. You have to reduce the plus lens for reading about one-half diopter. I tell the patient not to hold the print so close to the face, but farther away than the average person would. Oculists and opticians have a tendency to use large lenses. In this class of patients you must specify smaller lenses so that you can get the centers of the lenses a little within the pupillary centers of the eyes. This greatly relieves the strain on the convergence.

DR. C. D. WESCOTT, Chicago: In my practice I have been cautious about doing any operation on the eye muscles until every other means has been exhausted to make the patient comfortable. I hope Dr. Lemere does not wish to give us the impression that he has done the advancement or tucking operation without first attempting to relieve these cases by exercises. I have found that by painstaking refraction under cycloplegia, careful correction of the hyperphoria and training of the convergence I have been able to avoid many operations of this character. In the cases of absolute insufficiency we must operate, but the number in which we can get results by careful refraction and muscle-training is far in excess of those in which operation is needed.

DR. GEORGE H. PRICE, Nashville, Tenn.: Whether there are centers which control certain muscles is a question already decided for us by the physiologists and we have been working on this for some time, developing the exact conditions under which these functions are brought into action. If the original impulses in infancy were away from a direct line which should be established for normal physiologic function of any set of muscles, and if in trying to correct them we get into any little bypath, we have started wrong and it takes a long time to correct it. Deformities of the skull, spoken of by Dr. Risley, would account for this in a measure but I have seen all sorts of shapes and it does not necessarily mean that it determines the muscle condition of the eye. The centers are subject to education.

DR. H. B. LEMERE, Omaha, Neb.: In reply to Dr. Risley I may say that my whole endeavor has been to simplify matters rather than obscure them. I am proposing a simple practical solution of this problem. There are certain patients who, even after careful correction of refractive error under cycloplegia, still suffer from asthenopia. If this is due to insufficiency of convergence we always try, if practicable, to give proper exercises. This case illustrates the point: A boy aged 12, error +.50 D. sph. both eyes, unable to attend school. He was absolutely unable to converge nearer than 18 inches; distance orthophoria. Exercise could not be enforced. Operation gave good working convergence. I think that Dr. Savage's suggestion of a small candle for near exercise in convergence is excellent and will stimulate the convergence power better than the pencil ordinarily used. I thoroughly agree with Dr. Risley in considering many cases due to the altered relation between convergence and accommodation and this altered relation is always taken into consideration in the treatment. Dr. Wilkinson made a just criticism. I noticed the seeming discrepancy in the paper. The internal rectus,

However, after the handling of the operation may be assumed to have lost its spasm and any double vision would then indicate an over effect. Many opthalmologists would refuse to operate for insufficiency of convergence with esophoria in the distance and the patient would get no relief. I wish to state most emphatically that we do not operate unless we are satisfied that exercise will not correct the insufficiency. We do not rashly advise operation. The few patients on whom we operate could obtain relief from their prostrating symptoms in no other way. They should be relieved because if help is refused they go from pillar to post, have their glasses eternally changed, and become prey for quacks.

OCULAR VERTIGO

ALLEN GREENWOOD, M.D.
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Vertigo is produced by some disturbance in the functions of orientation and equilibration which results in a disturbed conception of our position in space, and is objective when objects about the patient seem to move, and subjective when patients feel themselves to be in motion.

Duel[1] says:

The function of orientation is a nice sense presided over by the vestibular apparatus; it is intimately associated with equilibration and brings this about by impressions through Deiters' nucleus to the oculomotor centers on one hand and to the motor neurons of the cord on the other.

Physiologists are divided into those who assert that all vertigo is essentially auditory in location, and those who believe that vertigo may result from disturbance of the cerebellum, entirely without the intervention of the vestibule and canals. The weight of opinion, however, seems to be in favor of the former view.

We know that the impulses conveyed from the eyes by way of muscle-controlling nerves are correlated in the cerebellum, and that in this way the function of equilibration is maintained; and we must remember that the portion of the vestibular nerve which arises in Deiters' nucleus communicates nerves to the nucleus of the motor nerves of the eye-muscles, so that labyrinthine abnormalities must have a disturbing influence on the ocular muscles. This intimate reflex association of the eye-muscles with the labyrinth has been carefully worked out by the otologists, to whom we are indebted for most of our knowledge concerning the influence of the aural apparatus on ocular movements.

Bartels[2] laid bare in the rabbit the externus and internus muscles, and after removal of the eyeballs and the remaining muscles, he determined the movement of these muscles during and after circulatory movements, and also after thermic stimulation of the ear. The curves so obtained showed how both the slow and rapid phases of nystagmus result from the contraction of one muscle contemporaneously with the perceptible relaxation of the antagonist. "These observations indicate the existence of a continuous tonic influence on the ocular musculature setting out from the ear."

Rapid rotation of the head, with a sudden stop, causes disturbances in the fluid of the semicircular canals, with a resulting vertigo and an accompanying nystagmus. Different varieties of the latter are produced according to which canal is most nearly horizontal, and thus its fluid most subject to the centrifugal force. These differences are obtained by holding the head of the person rotated erect or bent backward or far forward.

The thermic test also produces a vertigo with rotating nystagmus, with cold producing a rotation opposite to that of the ear experimented on, and heat the reverse.

For a complete study of the different forms of nystagmus produced by labyrinthine rotation and thermic tests, reference should be made to Scott's[3] tables.

In view of the foregoing may we not justly reason that a reverse reflex action can take place? That a particularly excessive and abnormally directed action of the ocular muscles, especially those concerned in ocular rotations, requires unusual and disturbing stimulation of the motor oculi nerves for its accomplishment is evident; and may not this unusual action of the motor oculi nerve centers cause reflexly, through Deiters' nucleus and the cerebellum, an unusual stimulation of the labyrinthine branches of the eighth nerve and thus cause vertigo? This ocular reflex stimulation of the nerve-endings in the labyrinth must ordinarily be much less than the stimulation produced by the rotation and thermic experiments, just as ocular vertigo is of a comparatively mild character, especially when compared with that symptom-complex known as Ménière's disease.

Davis[4] pointed out that several observers had mentioned that a rotatory vertigo and nystagmus may occur during convalescence in paresis of the eye-muscles, and says that "in such cases the mechanism of the vertigo and nystagmus may be plausibly explained by assuming that the labyrinth is reflexly influenced by the stimulation resulting from the innervation of the eighth nerve, communicated through Deiters' nucleus to the oculomotor muscles."

It is difficult for those who have not experienced severe attacks of vertigo, or been intimately associated with patients subject to such attacks, to appreciate the amount of distress and incapacity produced. In a nervous patient a nearly constant unsteadiness, or frequent attacks of dizziness, will incapacitate to a marked degree and result in actual phobias so that such patients will not venture from their rooms alone.

During recent years an increasing number of patients so afflicted, having tried in vain the aid of the internist and the otologist, have frequently turned to the ophthalmologist and found relief. Patients with vertigo without indications of aural, digestive or other disturbances, are now being sent to the ophthalmic surgeon, or voluntarily requesting his services, with the hope of relief from this occasional, but very distressing symptom of eye-strain. One writer states that eye-strain is the least frequent cause of vertigo; but this is evidently a fallacy due to the fact that ocular vertigo is often less severe than other forms, and is often cured by glasses at the presbyopic age, without the patients' having laid stress on the symptom, not believing it to be caused by their eyes. The future will probably show eye-strain to be one of the commoner causes.

About three years ago I became much interested in this subject and began tabulating my cases, and since that time I have seen nineteen in my private practice. Prior to that time I had, of course, seen cases of ocular vertigo; but not having kept special records in these cases, I have

1. Duel, A. B.: Abstract of discussion to article by Davis, George E., THE JOURNAL A. M. A., Oct. 8, 1910, p. 1277.
2. Bartels: Arch. f. Ophth. (Graefe's), lxxvii, 531.
3. Proc. Roy. Soc., April, 1909.
4. Davis, George E.: The Present Status of Vertigo Considered from a Diagnostic Standpoint, THE JOURNAL A. M. A., Oct. 8, 1910, p. 1272.

decided to report only those seen during the past three years, and in this small group of cases I have included only those in which the patients were sent by physicians, or came of their own volition, for the relief of vertigo. Of course, in many cases of asthenopia there will be a history of car-sickness or slight tendency to vertigo, though only as minor symptoms; but in all the cases, the histories of which I shall give below, marked vertigo was the chief or only symptom for the relief of which these patients consulted me, and all cases complicated by aural disturbances or marked internal diseases have been excluded; in fact, most of the patients had been previously examined by otologists and found free from aural defects, or had been treated by their family physicians for general disturbances.

That which influenced me to report these cases was the almost uniform type of refractive error which was found as the apparent cause of the vertigo, and the uniformly good results following the wearing of correcting-glasses.

CASE 1.—Miss S. M. F., aged 43, sent by her family physician because of a constant and annoying dizziness. The patient says that this is not sufficient ever to cause her to fall down, though she frequently has to catch hold of things to steady herself if she looks quickly from one place to another. The dizziness comes on in the morning after she has been up and about a very short time, and if she lies down and closes her eyes for a time it passes away. Examination of the eyes reveals the following: Right eye with +.75 sph. with +.62 cyl. ax. 100,° vision equals 20/20; left eye with +1. sph. with +.50 cyl. ax. 70,° vision equals 20/20. There is 1 degree of esophoria with the glasses on. The fundi are normal. These glasses were given for constant use, and she reports that since wearing them there has been no dizziness.

CASE 2.—Dr. E. E. H., aged 48, wearing the following bifocal glasses: +1.50 sph. with +1.00 sph. wafer added, complains of being dizzy most of the time, frequently with attacks of dizziness that incapacitate him, and at such times things seem to be whirling around. Dizziness quickly passes off when he lies down and closes his eyes. There are no signs of aural disturbance, and this physician was inclined to ascribe his dizziness to a beginning arteriosclerosis, and had made up his mind that treatment would probably avail him nothing. Examination of the eyes revealed: right eye with +2.50 sph. with +1.00 cyl. ax. 165,° vision equals 20/20; left eye with +3.00 sph. with +.50 cyl. ax. 180,° vision equals 20/20; +1.75 sph. added for near. Fundi normal, and with glasses he has orthophoria. Within forty-eight hours after these glasses had been put on, the dizziness had entirely disappeared, and during the past nine months there has been no sign of return.

CASE 3.—Mrs. F. A. B., aged 50, has a slight dizzy feeling practically all the time which commenced several years ago and has steadily grown a little more troublesome. She never has any bad dizzy spells and never has to hold on to things for support. Right eye +.50 sph. with +1.25 cyl. ax. 95,° vision equals 20/20; left eye +.87 sph. with +1.00 cyl. ax. 75,° vision equals 20/20. With these glasses on she has only 1 degree of exophoria. A +2.25 sph. was added to the above for the strength of the wafer. The fundi were normal. During the year that she has worn these glasses there has been no return of the vertigo.

CASE 4.—Miss R. P., aged 32, referred by her family physician for dizziness which is practically constant when she is up and about. By spells, things, as she expresses it, "whirl all around" her, so that she has to sit down or steady herself by some support. There are no evidences of any aural trouble. Right eye shows +.25 sph. with +.25 cyl. ax. 45°; left eye +.25 sph. with +.12 cyl. ax. 70.° With these glasses on she has absolute orthophoria. The fundi are normal. During the fifteen months that she has worn these glasses there has been no return of the dizziness.

CASE 5.—Mrs. E. M. B., aged 33, had been wearing glasses for two years, both eyes having a +2. sph. During the past three months there has been a steadily increasing feeling of dizziness and, as she expresses it, a constant feeling as though she were being pulled toward the right side. This is especially marked on any attempt to use her eyes, so that sometimes she is more comfortable if she closes the left eye entirely. Examination shows that the right eye will take a +4.00 sph. and the left eye a +4.25 sph. With these she gets a vision of 20/20, and has an esophoria of 4 degrees and left hyperphoria of 1 degree. She gets clearer vision with —.37 sph. added to the above. On account of the esophoria, however, she was given a slight overcorrection. During the several months that the patient has been wearing these glasses there has been no return of the dizziness, and she feels much better in every way. She has the same hyperphoria as before.

CASE 6.—G. A., a rather anemic girl of 11, came with the following history: She has been kept out of school for a number of months, on account of nearly constant dizziness, and during the past two months she has had three attacks in which she has fallen down apparently unconscious, and the family physician had made a diagnosis of epilepsy. Any attempt to use the eyes for close work causes them to ache and increases the dizziness. She is wearing a +1.00 sph. for both eyes which has not improved matters. After examination with a mydriatic the following glasses were prescribed: right eye +4.00 sph. with a +1.50 cyl. ax. 95°; left eye +3.75 sph. with +1.50 cyl. ax. 85.° She has orthophoria and no fundus trouble. The wearing of these glasses immediately stopped the dizziness and there have been no recurrences of the attacks for three years, except once or twice, when she has for some reason or other been without her glasses for several days.

CASE 7.—Miss E. F., aged 21, seen Jan. 2, 1913, with a history that evening study makes her eyes smart and burn; on looking around at such times she is apt to be very dizzy. Examination under a mydriatic gave right eye —.62 cyl. ax. 80°; left eye —.50 cyl. ax. 90.° Orthophoria and normal fundi. The wearing of these correcting-glasses has entirely eliminated the dizziness.

CASE 8.—Mr. A. F. M., aged 58, during the past two or three years has had increasingly frequent attacks of dizziness, and lately he waked up every morning with severe headache. There is no intranasal disturbance or sign of aural trouble, and no demonstrable arteriosclerosis. Right eye shows +.75 sph. with +.87 cyl. ax. 160°; left eye shows +.75 sph. with +.62 cyl. ax. 20°; takes +2.50 sph. added for correction of the presbyopia. Wearing these glasses has entirely prevented the headache and dizziness.

CASE 9.—Mrs. F. W., aged 54, for several years has been having gradually increasing attacks of dizziness accompanied by headache. She often wakes up with a pain in the back of her head which increases as the day advances and usually results in a good deal of vertigo, though she has never had it bad enough to render her unable to walk about. Examination of the right eye shows a —.75 cyl. at an axis of 5,° left eye —.75 cyl. at an axis of 15.° With these glasses she has a vision of 20/20 in both eyes. She takes a +2.25 sph. added for reading. No heterophorias. The fundi show a very slight arteriosclerosis, but the wearing of her distance glasses constantly, with reading-wafers, has entirely stopped dizziness during the past nine months.

CASE 10.—Mrs. J. M., aged 20, sent to me by her family physician with the hope that glasses would remove the vertigo which she has had for several months. Examination of the eyes shows +.25 cyl. ax. 90° in both eyes. The fundi were normal and the muscle balance good. The wearing of this slight correction for astigmatism, constantly, has entirely stopped the vertigo, and there has been no return of it during the past year and a half.

CASE 11.—Miss E. H., aged 32, sent by her family physician on account of a nearly constant, but slight dizziness. Examination of the eyes shows right eye +.25 cyl. ax. 50.° left eye +.25 cyl. ax. 120,° both eyes having a vision of 20/20. Fundi

normal and muscle balance normal. The wearing of these glasses has entirely relieved the patient of all symptoms of dizziness during the past two years.

CASE 12.—Mr. A. C., aged 36, sent by his family physician with the report that for several months any use of his eyes had produced discomfort ending in nausea and dizziness. Right eye shows +.25 cyl. ax. 30.° vision 20/20; left eye —.37 sph. with +.75 cyl. ax. 105.° vision 20/20. Fundi normal and no heterophorias. The wearing of these glasses has entirely removed all signs of the previous trouble.

CASE 13.—Mrs. G. S., aged 29, for the past year and a half has been very dizzy some part of nearly every day. This dizziness has been increasing so that at present she is dizzy most of the time, though not to such an extent as to incapacitate her. She is especially dizzy after any attempt to concentrate her vision, and at such times her eyes feel strained, but she has no headache. Examination shows the right eye +.37 cyl. ax. 135,° and left eye +.87 cyl. ax. 85.° For the two years that the patient has been wearing these glasses there has been no return of dizziness. In this case there are no signs of muscle trouble and nothing to be seen in the fundi.

CASE 14.—Mrs. J. C., aged 39, has been subject to headache and dizziness ever since she was a little child. Frequently when looking quickly from one object to another, she becomes so dizzy that she has to hold on to things to steady herself. Examination shows right eye +.50 sph. with +1.00 cyl. ax. 180°; left eye +.50 sph. with +1.50 cyl. ax. 175.° Fundi normal and muscle balance good.

CASE 15.—Miss E. M., aged 22, has been growing nearsighted for several years until at present it is difficult for her to see car signs clearly. The last year or two she has been having increasingly frequent attacks of headache and dizziness. Examination shows right eye —.75 sph. with —.25 cyl. ax. 70°; left eye —1.12 sph. with —.25 cyl. ax. 110.° Fundi are normal and muscle test shows orthophoria with these glasses on. During the three years she has worn these glasses there has been no return of the dizziness.

CASE 16.—Miss S. M., aged 24, for several years has done fine watch work, and during that time has had increasingly frequent attacks of dizziness and has a slight amount of vertigo all of the time while at her work. The patient has never worn glasses. Right eye shows +.75 sph. with +3.25 cyl. ax. 60.° vision equals 20/20; left eye +3.75 cyl. ax. 95,° vision equals 20/20. Fundi normal and muscle balance good. As soon as these glasses were put on the dizziness almost immediately disappeared, and during the year that she has worn them there has been no return.

CASE 17.—Mr. H. E., aged 40, has for several years been subject to spells of dizziness, and at times he has to stand still for a moment before he dares to move ahead. This he finds is especially so when entering a light room from a dark. Right eye —.25 sph. with —.37 cyl. ax. 150.° vision equals 20/20; left eye +.50 sph. with —.62 cyl. ax. 105.° vision equals 20/20. Muscle balance and fundi normal.

CASE 18.—Mr. W. C., aged 56, greatly troubled by nausea and dizziness, particularly when walking about. Moving objects almost always make him dizzy. Right eye +1.00 sph. vision equals 20/20; left eye +.75 sph. with +.25 cyl. ax. 105.° vision equals 20/20. Has 1 degree of exophoria, and the fundi are normal.

CASE 19.—Miss E. C. D., aged 53, sent by her physician, who himself furnished Case 2 of this series. She has a great deal of dizziness, is unable to turn her head and look about without becoming dizzy, and at times has nausea and vomiting lasting several hours. O. D. +.62 cyl. ax. 80°; O. S. +.50 cyl. ax. 100.° No fundus trouble; with these glasses the patient has 0.5 degree of esophoria; +2.25 sph. was added for bifocal, and after wearing the glasses several months the patient reported no return of dizziness or nausea.

A study of these cases shows that out of the nineteen patients there were seventeen having astigmatic eyes, with one or both axes oblique, fifteen showing asymmetric axes, and seventeen with slight anisometropia. Only one case had an insufficiently corrected simple hypermetropia of high grade, and this one case was also the only one showing a hyperphoria; but in this case the correction of the hypermetropia caused the vertigo to disappear, with the hyperphoria still remaining.

We all know that cases of vertigo result from muscular palsies, as painted out by Davis[4] and Croft;[5] but I have personally seen none since I began tabulating my cases. High grades of heterophoria are also considered to be the cause of a certain number of cases, but during the period mentioned previously none of these has come under my observation, though I have seen a good many patients with marked heterophorias and have questioned some of them in regard to dizziness without eliciting any history of vertigo worthy of notice. In view of this I feel justified in stating that in all probability the majority of the cases of ocular vertigo result from moderate errors of refraction, and usually occur in patients whose eyes show astigmatism with asymmetric oblique axes.

In other words, those patients whose eyes, when used, cause head-tiltings and disturbances to the normal ocular rotations and movements may produce vertigo from reflex irritations of the labyrinthine nerves the reverse of those conditions of rotary nystagmus produced reflexly by labyrinthine disturbances.

I find that most of the reported cases of ocular vertigo have occurred, as have mine, in patients with astigmatism with oblique axes, including one of Dr. Dixon's[6] cases, in which the patient, who had no astigmatism, was wearing +.75 cylinders placed at oblique and asymmetric axes; the removal of these glasses probably had as much to do with the cure of the vertigo as the wearing of the proper hypermetropic correction.

Croft[5], in his article on ocular vertigo, mentions ocular-muscle palsies as occasionally the cause of vertigo and also possibly heterophorias, but ascribes the majority of cases to errors of refraction.

As the result of our experience we all are frequently warning patients for whom we have prescribed cylindrical glasses with axes oblique to expect some distortion of images and disturbances of orientation, and probably resulting vertigo. It often happens that such patients have to get used to their glasses very gradually owing to this. Minus cylinders, at oblique but nearly horizontal axes, will cause a receding and tipping of images looked at, such as the floor or curb-stone, while plus cylinders, so placed, will produce the reverse appearance, and the effort on the part of the eyes to overcome this difficulty of orientation produces the vertigo. The following case illustrates this well:

Mrs. B., aged 39, was given O. D. —.37 sph. with +1.50 cyl. ax. 45°; O. S. —.25 sph. with +1.00 cyl. ax. 135.° Fundi and muscle balance normal. With these glasses she obtained great relief when using them for reading, but the minute she attempted to look about the room, or walk about, she experienced extreme vertigo, so that it was impossible for her to wear the glasses constantly at first.

I have seen + .25 cylinders at axes slightly above the horizontal produce just as marked vertigo as seen in this case, and I find that when patients become accustomed to such glasses the removal of them will often cause temporary vertigo.

5. Croft: Boston Med. and Surg. Jour., September, 1905.
6. Dixon: Ophth. Rec., March, 1907.

The accurate adjustment of the eyes, by normally acting ocular muscles and fusion centers, is essential for binocular vision, judgment of distances, and especially perspective. Eyes having astigmatism with the meridians of greatest and least curves oblique, and eyes having cylinders with axes oblique placed in front of them have this perspective disturbed, and the resulting unnatural activity required of the oculomoter nerves and the higher fusion centers may cause reflex vertigo or possibly vertigo independent of the labyrinths.

ABSTRACT OF DISCUSSION

DR. ALBERT E. BULSON, JR., Fort Wayne, Ind.: In a study of the subject of vertigo it should be remembered that the labyrinth is not the only source of impulses which help to maintain equilibrium as evidenced by the fact that destruction of the labyrinths produces only temporary loss of balance. Braun and Friesner, in a recent work, say that the other sources of impulses for the preservation of our equilibrium perform the function of the labyrinth vicariously, or what is more probable, the centers in which all these impulses are gathered accommodate themselves to the loss of the normal influence of the labyrinth, and a compensation results. Just how the impulses from the labyrinths share with those from other sources in maintaining our balance, is a difficult matter to determine, and in the present state of our knowledge is largely speculative. Without doubt, however, all of these centripetal impulses combine harmoniously and partly reflexly, at all events automatically, to establish our balance without our realization of the processes involved. Every disharmony in these impulses which serve to maintain our equilibrium causes, if sufficiently pronounced, a marked disturbance in balance. The labyrinth is probably always active, in the sense that it constantly sends to the centers impulses acquainting us with our position in space, and also with the changes in our position. Under ordinary circumstances reflex stimuli are constantly aroused with perceptions of our position and its changes, whose purpose it is to cause the eye muscles and the body muscles to accommodate themselves to the conditions that call forth these perceptions. It is an established fact that the labyrinths are in relation with the central nuclei of the muscles of both eyes and a powerful impulse aroused in one labyrinth, even after the complete destruction of the opposite labyrinth, is capable of causing both eyes to execute nystagmic movements in all planes, and directed either to the sound side or to the other. All the centripetal impulses, whose purpose it is to maintain our equilibrium, are gathered together in the cerebellum, so that the latter may be considered, in a sense, the center for the labyrinth. There probably is no single, definite center on which all of the centripetal impulses exert their influences. The cerebellum receives centripetal impulses from the labyrinth, sensory impulses from the muscles and joints, and visual impulses from the eyes and it sends out to the muscles of the head, trunk and extremities motor impulses, the purpose of which is to maintain the equilibrium of the body. In the cerebellar cortex the motor impulses meet the centripetal impulses from the labyrinth, the muscles, the joints and the eyes, and are there acted on in such a way as to become effective in maintaining the equilibrium of the body Vertigo, in itself, is of little value in determining the nature of the underlying affection, for, as stated by Mills, irritation may be reflected to the apparatus of equilibration from almost any part of the body, near or far. It is plausible, therefore, to assume that ocular vertigo, which is reflex, may, as pointed out by Dr. Greenwood, be a reverse action in which the irritation produced by accommodation strain and muscular insufficiencies, may produce an abnormal stimulus on a more or less non-resisting equilibratory apparatus in which the disharmony is manifest in either the labyrinth or cerebellum. Posey states that any uncorrected error of refraction, presbyopia or heterophoria, in an individual predisposed to nervous affections, may be responsible for an ocular vertigo and he quotes observers who state that vertigo occurs in about 12 per cent. of all cases of asthenopia, including both accommodative and muscular, in which no disease of the visual apparatus can be shown. It is common also in those who have asthenopia from wearing inaccurately adapted lenses, either in the strength of the lens or in the adjustment of the axis of the cylinder. Posey refers to the joint papers of Thomson and Weir Mitchell, who point out that vertigo may be a symptom of eye-strain, a fact that is being verified in the routine work of probably all oculists. In my own experience the cases of ocular vertigo, which are relatively frequent, have usually occurred in patients who belong to the type known as neurotic, or whose resisting power has been lowered by disease, overwork, dissipation or failure to observe the ordinary rules of hygienic living. The vertigo is usually associated with one or more other nervous symptoms, including headaches, nausea, irritability, etc. The attacks are brought on by eye-use and it is my experience that they occur as frequently under accommodative effort for distance vision as they do for near vision. The attacks are frequently preceded by headaches, nervous irritability or a sense of weariness or oppression. Usually the amount of error is small and almost invariably it is of the astigmatic type with axes off from the vertical or horizontal planes. The existence of slight heterophorias has seemingly had little influence in the production of the vertigo, inasmuch as the correction of refraction in the majority of cases has been sufficient to relieve the vertigo, although occasionally a prism, more often to correct a hyperphoria, has been required. Ocular vertigo is invariably relieved by cycloplegia, or by closing the eyes for a time, either while sitting or lying down, and this forms a valuable diagnostic indication as to the kind of vertigo we are dealing with. It should be noticed that practically identical errors will produce no symptoms in some persons whereas in others they will produce the most distressing symptoms. The reason for this is easily explained by the lack of nerve balance possessed by a class of patients, whom, for want of a better term, we call neurotics. The recognition of an uncorrected error of refraction, often seemingly of little consequence as the etiologic factor in the production of some distressing forms of vertigo, is deserving of the emphasis placed on the subject by Dr. Greenwood.

DR. G. C. SAVAGE, Nashville, Tenn.: Dr. Greenwood has not given too much emphasis to the fact that an oblique astigmatism produces vertigo. It is also capable of producing all the other symptoms that come from astigmatism which is not oblique. Lenses correcting the oblique astigmatism ultimately bring relief from the vertigo and the other symptoms caused by the astigmatism. The lenses given for the correction of astigmatism which is not oblique gives perfect satisfaction at once, but the lenses correcting oblique astigmatism are always a source of peculiar annoyance for a greater or less length of time. Oblique astigmatism, with the meridians of greatest curvature diverging above, whether the astigmatism be myopic or hyperopic, so long as not corrected, causes the superior oblique muscles to tort the eyes inward for the purpose of harmonizing distorted images. This action on the part of the superior oblique muscles does not cease at once when the correcting cylinders are given, but the time required to become accustomed to such lenses is usually very short. In all cases of oblique astigmatism with the meridians of greatest curvature converging above, whether the astigmatism is myopic or hyperopic, so long as uncorrected, it compels the inferior oblique muscles to tort the eyes outward, in order to harmonize the distorted images. The correcting cylinders at once relieve the distortion of the images but the old habit of action on the part of the inferior obliques continues for a considerable time in nearly all cases and so long as this habit continues the patient is annoyed by the false appearance of things. In the former class of cases the patient will have to go up hill, for a level surface will be slanting towards him, but in the latter class of cases the patient will have to go down hill, for a level surface will be slanting from him. The first class of patients will become accustomed to their glasses much sooner than the second class. A few days' annoyance will cover the period for the first class, but the annoyance

for the others will continue for a week and sometimes much longer.

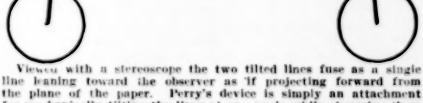
DR. E. L. JONES, Cumberland, Md.: Vertigo caused by the eyes has impressed me as one of the commonest symptoms. I have had a number of little children, who had not reached the stage of headaches, complain of giddiness and if anything comes up that directs attention to the eyes it is corrected. I have seen old people in the sixties who had extreme giddiness which good physicians had tried to relieve. Apparently their eyes would be all right, they might have been wearing presbyopic lenses for a quarter of a century. The slightest error in refraction may produce it. We often find a low grade of astigmatism that does not blur the vision in the slightest. The axes have been inclined and the wearing of glasses has brought marvelous relief. I recall the case, ten or twelve years ago, of a stonecutter who had extreme dizziness and who was not dissipated or neurotic. He could work only a day or two at a time. His eyes were refracted under complete cycloplegia. He had 1 diopter spherical of hyperopia. It was corrected and after he became used to the glasses he had no further vertigo. The explanation of vertigo is varying. In one instance a man had been in bed about a week. He said he could not come to the office. When his eyes were tested he could sit up only about five minutes at a time. He had a small vertical astigmatism and in about three or four weeks had recovered from the vertigo. I have seen many cases of men disabled from their vocations on account of vertigo who had no ocular symptoms whatever, no distressing symptoms about the eyes, no particular reflexes at all, no headaches or what are considered the ordinary symptoms of eye-strain. I keep fairly accurate records of the cases I do under cycloplegia. In 1912 I found 168 cases of manifest reflex ocular symptoms, of whom 69 complained of vertigo of varying degrees. Of 40 men, 15 had vertigo; of 96 women, 42 had it; of 15 boys, 7 had it, and of 17 girls, 5 had it. I am not very reverential in the presence of the heterophorias but I take off my hat to astigmatism, especially if it has a tilted angle.

DR. LEWIS H. TAYLOR, Wilkes-Barre, Pa.: Dr. Bulson speaks of the fact that these patients are of the neurotic type. Perhaps the majority of them are neurotic because of the astigmatism. In the beginning they are not neurotic. I recall particularly one of the surprises of my early practice. A physician who was approaching a condition of melancholy, being on the verge of collapse, came to me and said he was worried and was unable to go on with his work. All that was done was to correct his astigmatism, which was of low degree. As I remember it, this man had selected very low cylinders, but the axes were 15 in each eye. The correction restored the man to health and he is hale and hearty, a man past 70, still in active practice. This occurred twenty years ago. Vertigo was a prominent symptom in the case of another physician who was in much the same condition, ready for a breakdown. The result of the correction was the same. These are only two cases of many that I can recall. It seems to me that in these cases of vertigo we should always proceed to correct the refractive error, and if we do not succeed in correcting the vertigo we will find other trouble, probably in the labyrinth.

DR. C. D. WESCOTT, Chicago: These patients are not always neurotic. A mechanic, not engaged in fine work, was referred to me after passing careful examinations by an internist and an aurist. I found less than a diopter of astigmatism, with oblique axes, and he was immediately relieved by the use of his correction. A few months afterward I was disappointed to see him return with the original symptoms and thought possibly I had misjudged his case. Full cycloplegia showed that during the interval there had been a slight change in the axes of his astigmatism which I corrected and he was promptly relieved again.

DR. F. PARK LEWIS, Buffalo, N. Y.: I think we have all had experience with the various ocular vertigos, but two or three years ago I brought to the attention of the profession, before the Academy of Medicine in Cleveland, conditions associated with this vertigo and the relation of vertigo with or without astigmatism, to aural conditions, such as tinnitus, etc. I found a paper by Rolleau in which he called attention to various conditions associated with eye-strain. After having presented my paper Dr. Risley discussed it and alluded to the fact that a variety of conditions were associated with eye-strain. The next year Dr. Theobald presented a paper before this section in which he quoted my findings and added still others. At least four of our colleagues took up the question of the relationship between the eyes and various aural conditions. That fact having been called to the attention of a colleague, he relieved a case of tinnitus by correcting the refraction. This condition is not widely enough known and I intend to collect the facts bearing on the direct relationship between the eye and the ear. Whether it is through Deiter's nucleus or otherwise, there is direct connection between binocular vision and aural conditions and I shall be glad to have a note from you as to your experience along this line.

DR. WALTER B. LANCASTER, Boston: It is interesting to theorize as to the possible mode of operation by which oblique astigmatism may produce vertigo. We maintain our sense of equilibrium by afferent messages to the higher centers from many sources such as the semicircular canals, the visual apparatus, the muscle sense, the tactile sense, superficial and deep. All these messages are intimately correlated by an adequate system of connecting paths and presided over by centers of which we may mention the important one in the cerebellum. Like all important functions this has a wide margin of safety in normal individuals. I mean by this that there can be disturbances in various parts of this mechanism without throwing it out of gear so far as to cause vertigo. A disturbance anywhere in the mechanism of equilibrium may cause vertigo in a patient who has little margin of safety to spare, even though an equal disturbance in this mech-



Viewed with a stereoscope the two tilted lines fuse as a single line leaning toward the observer as if projecting forward from the plane of the paper. Perry's device is simply an attachment for mechanically tilting the lines at any angle while observing them with the stereoscope.

anism in a more normal subject would not cause vertigo but only a slight disagreeable sensation. That is how I account for the fact that of the thousands who have oblique astigmatism only a few have vertigo of sufficient degree to seek medical advice. The patient must be susceptible. The nervous system, in that particular regard, must be sufficiently unstable to be deranged by so slight a cause, if indeed it be a slight cause that makes lines we had reason to think were vertical appear slanting and makes other objects occupy correspondingly abnormal and unnatural positions. It is well known that oblique cylinders and oblique astigmatism cause vertical lines to appear slanting. Savage has written much on this subject. The common explanation of how these slanting lines are fused and not seen double is that the eyes are rotated to correspond to the slant by the process called torsion or declination but this is an error. A stereoscopic toy slightly modified from the one of Perry, which he published in 1895 to show that the eyes do undergo torsion, I use to disprove it. By moving the handle up and down you can imitate the slanting of the vertical lines which would be produced by oblique cylinders. The lines are fused, not by torsion, but by virtue of what I call the function of *stereoscopic fusion for relief* or depth; Verhoeff calls it "psychical compensation." This makes the fused line appear to extend forward or backward from the surface of the paper, out into space, giving the sense of depth, relief, third dimension of space. It is not a function of the retina but of the visual centers of the brain and is no doubt closely associated, as

Dr. Greenwood points out, by connecting paths with the other centers concerned in the perception of our relation to objects in space — the function of orientation and equilibrium.

DR. GEORGE S. DERBY, Boston: I should like to ask Dr. Greenwood whether he makes any distinction between vertigo and dizziness. I agree with Dr. Bulson that many of these cases are neurotic and I feel a great deal of doubt as to whether such small degrees of refractive error would cause dizziness, as in Cases 3 and 10. I take exception to Dr. Greenwood's statement in Case 10. He says: "The wearing of this slight correction of the astigmatism constantly has entirely stopped the vertigo." The lens was a plus 0.25 cyl. ax. 9 in both eyes. I think it is difficult to say that the wearing of that correction corrected the astigmatism and stopped the vertigo alone. I call attention to the fact that the prescribing of glasses is one of the most powerful suggestive methods of treatment.

DR. EDWARD JACKSON, Denver: A physician of 50 years, weighing 250 pounds, successful in his practice, well-to-do, and a man who is not in the ordinary sense neurotic, came to me for persistent vertigo, for which he had tried all sorts of things. He required 0.25 and 0.37 cylinders, which relieved him. In two or three months he thought he was well and left off the glasses. The vertigo returned. He put them on again and wore them for over two years. Then he found that he got along pretty well without them for several months, and the vertigo gradually returned. I remeasured the eyes and found about the same amount of astigmatism. He put on glasses and again obtained relief. I think that very low degrees of ametropia may cause perceptible and annoying vertigo.

DR. HIRAM WOODS, Baltimore: There are two possible errors in diagnosis in low grades of astigmatism. Some years ago Dr. Theobald of Baltimore called attention to the fact that the normal near balance is exophoria of from 2 to 5 degrees, as compared with the distant balance. For instance, if there is orthophoria in distance we must look for a near balance of from 2 to 5 degrees of exophoria. There has been considerable discussion as to whether or not this is true, but I believe it is, and that such a relation should be sought. We get a class of cases in which the near balance is esophoric when compared with the distance balance. Theobald asserts that when this exists, after correction of refraction error, the meaning is ciliary asthenopia. The accommodative muscle demands excessive innervation; positive relative convergence is low; that is, there is a very low adduction, the patient being unable to separate his Ac. and Con. Hence, the excessive innervation goes to the internal recti as well as to the ciliary muscles, and an apparent near esophoria results. It is to be treated by convex glasses for near. If every vestige of static error is corrected there is, I think, no doubt of the truth of Theobald's contention, but one can easily fall into error with these low grades of astigmatism. The ametropia is slight and accommodation corrects it with normal vision, but this is done by excessive ciliary action. Hence, there is corresponding overaction of the interni and apparent near esophoria. One diagnostic error is failure to find the low astigmatism. The presence of normal vision misleads. The clue to the condition is variations from day to day in subjective tests under cycloplegia. In other words, the eye does not yield readily to cycloplegics. Then there is liable to be a reddened disk, significant of chorioidal irritation. When a young patient presents the symptoms outlined by Dr. Greenwood and has, before the use of a cycloplegic, a near relative esophoric balance, I think we ought to push the cycloplegic until several subjective examinations agree with each other and with objective measurements. In this way we relieve ciliary irritation. If we do not uncover these low grades of astigmatism, the patient cannot get well, and will soon present seeming need for plus glasses in the near. The other diagnostic pitfall is interpreting this near esophoric balance as real esophoria and treating it with prisms, bases out. The guide is assured cycloplegia. Just one illustrative case: A young woman was wearing + 1.37 spherical for near vision. Her distance balance was orthophoric, her near, esophoric without glasses, exophoric with them. Under scopolamin there was inconsistency between examinations for several days. Finally, it settled down to low hypermetropic astigmatism with axes at 30° and 150°. Under these, the apparent esophoria in the near disappeared, and the normal near exophoria took its place. In my judgment there is no class of refraction cases so apt to mislead one and so important to the patient.

DR. ALLEN GREENWOOD, Boston: The case Dr. Derby mentions was the only astigmatic one in which the axes were not somewhat tipped and yet the patient was sent to me after having been through the hands of the family physician and an aurist for a dizziness which had been very distressing for two or three months. For a year and a half there has been no vertigo. It may be mental impression but the dizziness has disappeared. In other cases with equally slight astigmatism in which the axes were oblique, taking off the glasses has reproduced the dizziness, so I do not believe that in all slight cases this relief has been due to mental effect. I have used vertigo and dizziness as synonymous terms. I have seen many cases, during the past years, of slight vertigo along with other symptoms, particularly headache, and I have seen a great deal of car sickness which is somewhat akin to this condition, but all the cases in this respect have come to me for the relief of marked dizziness. I have not found that these patients were especially neurotic, although some of them were. One of my house officers was actually unconscious for a few minutes after a rotation test and this recalls the case of the girl who was thought to be epileptic but has had no attacks since wearing glasses. The physician I referred to was a man who weighed 200 pounds and was in splendid condition. He was ready to give up his practice for what he supposed was arteriosclerosis. For the last two or three years he has been comfortable and free from dizziness and able to carry on a large practice.

IS THE PERCENTAGE OF MYOPIC EYES DIMINISHING?

SAMUEL D. RISLEY, M.D.
PHILADELPHIA

In my report of the examinations of the eyes of the schoolchildren in Philadelphia, published in 1881, it was shown that the increasing percentage of myopia in the schools as the pupils advanced in age and school progress, as was set forth by the statistics of many observers both in Europe and America, was due not so much to the want of hygienic precautions in the schools as to the existing congenital defects in the eyes of the children. Many examples, it is true, were presented showing the baneful influence of inadequately lighted schoolrooms and other faulty environments over the eyes of all young children, but it was obvious from their collated statistics that the crux of the matter lay in the congenital visual defects. The findings of the Philadelphia examinations were set forth in elaborate statistical tables, and graphically displayed in a series of percentage curves, the latter of which are here reproduced for reference (Charts 1, 2, 3 and 4).

The entire work was summarized[1] in the following conclusion and recommendation:

That, *given an emmetropic or normal eye, the probabilities are that no harm will come to it from the educational process.* On the other hand, *given an eye with an anomaly of refraction, especially astigmatism, the probabilities are, other things being equal, that the educational process will be fraught with pain and danger to the eye.*

1. Tr. Med. Soc. of Pennsylvania, 1881.

Therefore, that before entering school the possible presence of defects of vision should be excluded, and any existing error of refraction corrected as soon as any trouble is experienced.

Briefly stated, it was shown by this investigation that the emmetropic eye, notwithstanding its relatively small percentage of the whole number, was to be regarded as the model or standard eye, since it passed through the stress and strain of school life with a minimum of pain and peril, and maintained a nearly uniform percentage through all the years of school life; that pain, lowered acuity of vision and disease steadily increased as age and school progress advanced in the eyes with defects of refraction, especially in astigmatic eyes; that with increasing school age the percentage of eyes with hypermetropic refraction steadily fell, while the percentage of eyes with myopic refraction increased in a corresponding ratio, the increasing refraction being associated with a steadily increasing percentage of pain, lowered acuity of vision and disease of the intra-ocular tunics. As a corollary growing out of these findings and conclusions it was recommended that no child should be permitted to enter on the educational process until the eyes had been examined.

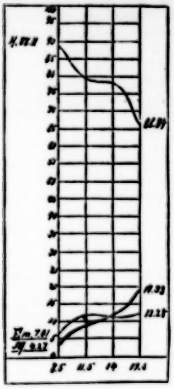


Chart 1

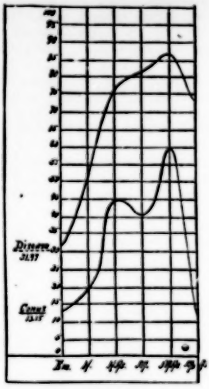


Chart 2

Chart 1.—Steady rise in the percentage of myopia as the school age advanced from 8½ to 17½ years and a corresponding fall in the percentage of hypermetropic eyes.

Chart 2.—Disease in relation to refraction.

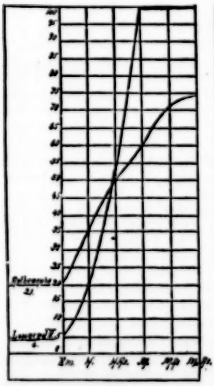


Chart 3

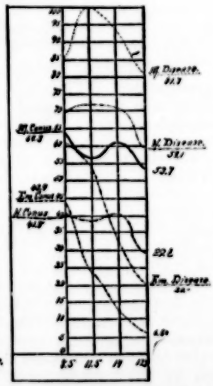


Chart 4

Chart 3.—Vision and pain in relation to refraction.
Chart 4.—Disease in relation to age.

While these statistics were in course of preparation I published,[2] and later presented before the American Ophthalmological Society, the history of a large series of cases,[3] all taken from my private case-books, in which the eyes had passed while under observation from hypermetropic to myopic refraction through the *turnstile of astigmatism*. These cases were presented and published to fortify the claims made in the report of the school examinations.

Twelve years later, probably because of this work, I was requested by the late Dr. William F. Norris to write the article on "School Hygiene" which appeared in 1894 in the second volume of Norris and Oliver's "A System of Diseases of the Eye." While preparing this article it occurred to me that if the conclusions based on the school report and published in 1881 were true, some demonstration of their truth should be shown after twenty years in a careful analysis of all cases applying in the routine of professional work for relief from asthenopia by the careful correction of existing congenital errors of refraction. In Philadelphia during the period from 1874 to 1893, inclusive, ophthalmic surgeons had habitually ordered glasses for the relief of asthenopia. During this period many cases of rebellious headache and nerve storms, after Dr. S. Weir Mitchell's famous paper on their relation to eye-strain, were referred to the ophthalmic surgeon for consultation and treatment. It was therefore logical, reasoning *a priori*,

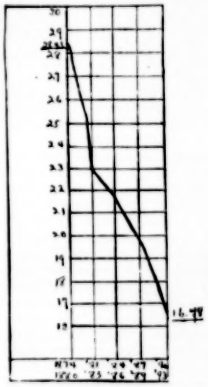


Chart 5

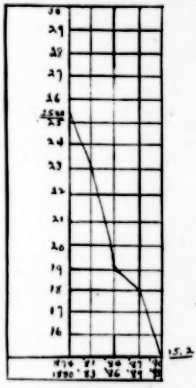


Chart 6

Chart 5.—Curve showing the steady fall in the percentage and degree of myopia from 1874 to 1893, inclusive, constructed from Table 1. From private case books.

Chart 6.—Curve showing the steady fall in the percentage and degree of myopia from 1874 to 1893, inclusive, constructed from Table 1. From opticians' books.

2. Risley, Samuel D.: Am. Jour. Med. Sc., October, 1880, p. 442.
3. Risley, Samuel D.: Tr. Am. Ophth. Soc., 1884, 1887.

to assume, if the conclusions of the school report based on the statistics there formulated were true, that such an analysis of the cases as was proposed should show diminishing percentage in the relative number of myopic eyes and a lower degree of myopia. The work was undertaken, and the resulting figures were based on the refraction as shown by the glasses ordered for distance and constant wear. The work covered the correcting-glasses for a total of 195,754 eyes, of which 8,736 were from my private case-books, all of the latter having been painstaking corrections under the use of mydriatics, pressed to the thorough paralysis of the accommodation.

The percentage charts and the tables on which they are based are here reproduced for comparison with those to be presented in the present study:

This paper is a continuation of the study from 1894 to 1912, inclusive. The analysis is made only from my private case-books and mydriatic corrections, the mydriatic being pressed to complete cycloplegia, and a painstaking correction of the astigmatism and careful study of the binocular balance. The total number of eyes falling under the strict requirements of the analysis is 6,850.

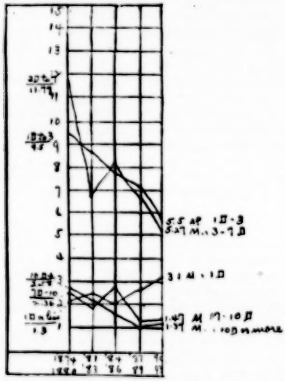


Chart 7. Curves showing the steady fall in the percentage and degree of myopia from 1874 to 1893, inclusive, constructed from Table 1. From private case books.

At the present time it has been found possible to complete the analysis only for the group of cases in the first three years, 1894 to 1896, inclusive, and for the group in the last three years, 1910, 1911 and 1912, giving for these years a total of 2,297 eyes. In all of these, painstaking effort was made by every means at our command to determine the static refraction of each eye, the determination being made after the more or less prolonged use of the stronger mydriatics. Homatropin was used only for elderly persons, and then for its therapeutic effects over irritable and inflamed eyes.

Many features of great interest and importance have been omitted from the report. For example, had the design been to show the significant and important anatomic relationship between anomalies of refraction and the abnormalities of binocular balance as etiologic factors in asthenopia and intra-ocular disease, a vast fund of information could have been set forth, but would have required the presentation of a large number of case histories by way of illustration and demonstration. Had this fallen within the scope of the paper, a large group of patients in which hypermetropic eyes had passed into myopic refraction *through the turnstile of astigmatism* would have been presented. They would not have been new cases, however, that is, patients coming for the first time for advice, but old patients who for some reason had neglected the correcting-glasses originally given, or as the result of ill-health, uveal disease, etc., had suffered an increase of refraction in their eyes.

TABLE 1.—PERCENTAGE OF MYOPIC EYES, 1874-1893

Years	Number of Eyes	Number of Myopic Eyes	Percentage of Myopia	Percentage of Simple M.	Percentage of Myopic Astigmatism	M. = more than 10 D.	M. = 7 to 10 D.	M. = 3 to 7 D.	M. = 1 to 3 D.	M. = 1 D or less
1874-1881	1,956	559	28.43	15.5	85.5	2.58	2.36	11.77	9.5	2.3
1881-1883	1,354	308	23.05	15.45	84.55	2.10	1.96	6.92	8.6	2.3
1883-1886	1,376	313	22.07	5.75	94.25	1.67	2.90	8.43	7.8	1.9
1886-1889	2,154	427	19.82	6.50	93.50	1.10	1.20	6.70	7.5	2.7
1889-1893	1,896	318	16.78	5.66	94.34	1.37	1.47	5.27	5.5	3.1
Totals and averages	8,736	1,925	22.03	9.67	90.33	1.76	1.98	7.84	7.78	2.46

In Table 3 the plan has been made to conform as closely as possible to that presented in the paper on "School Hygiene" in 1894 (Table 1). By reference to these tables and the accompanying percentage charts it will be seen that the percentage of myopic eyes beginning at 28.43 per cent. in 1874 rapidly fell through the intervening years to 16.78 per cent. in 1893, the average percentage for twenty years being 22.03.

TABLE 2.—PERCENTAGE OF MYOPIC EYES, 1894-1896 AND 1910-1912

Years	Number of Eyes	Number of Myopic Eyes	Percentage of Myopia	M. = More than 10 D.	M. = 7 to 10 D.	M. = 3 to 7 D.	M. = 1 to 3 D.	M. = 1 D. or less
1894	498	75	15.06	5.33	10.66	26.66	26.66	29.2
1895	440	57	13.40	7.02	7.02	17.54	21.05	40.35
1896	483	66	13.86	13.48	3.03	22.73	37.88	22.73
Totals and averages	1,421	198	14.07	8.61	6.90	22.31	28.53	30.76
1910	275	37	13.45	.....	5.41	45.95	18.91	29.73
1911	292	24	8.21	.....	.....	12.50	41.67	45.83
1912	309	37	11.97	5.41	2.70	21.62	37.84	32.43
Totals and averages	876	98	11.21	1.80	2.70	26.69	32.80	35.99

In the tables now presented (Tables 2 and 3) it will be seen that the percentage of myopic eyes for 1904 had fallen to 15.06 per cent., for 1905 to 13.4, and for 1906 to 13.86, giving an average for the three years of 14.07 per cent., as compared to 16.78 for the preceding three years, or 22.03 per cent., the average for the preceding twenty years. The diminishing percentage of myopia is made strikingly manifest by the figures shown in the last three years, 1910 to 1912, inclusive. In 1910 there was 13.45 per cent. of myopic eyes, which fell in 1911 to 8.21 per cent., but rose in 1912 to 11.97 per cent., or an average for the three years of 11.21 per cent. The result for the entire period

from 1874 to 1912 is graphically set forth in Chart 9, in which the average percentage of myopia for each succeeding period of three years is shown by the steadily falling curve from 28.43 per cent. in 1874 to 11.21 per cent. in 1912.

In Table 3 (not included in the presessional volume), the percentage of the degrees of myopia from 1 D. or less to 10 D. or greater are here calculated on the total number of eyes, as in Table 1 from "School Hygiene," whereas in Table 2 these percentages were calculated on the total number of myopic eyes.

TABLE 3.—SHOWING DIMINISHING PERCENTAGE OF MYOPIC EYES AND A FALL IN THE DEGREE OF THE MYOPIA

Years.	No. of Eyes	No. of Myopic Eyes	Percentage of Myopia	M.=10 D. or more	M.=7 to 10 D.	M.=3 to 7 D.	M.=1 to 3 D.	M.=less than 1 D.
1894	498	75	15.00	.80	1.00	4.12	4.12	4.12
1895	440	57	13.40	.91	.91	2.27	2.73	5.23
1896	483	66	13.86	1.86	.41	3.10	5.18	3.10
Totals and averages ..	1,421	198	14.07	1.19	.97	3.16	4.01	4.25
1910	278	37	13.45	.....	.73	6.18	2.54	4.00
1911	292	24	8.21	.....	.....	1.3	3.42	3.73
1912	309	37	11.97	.61	.32	2.59	4.56	3.88
Totals and averages ...	876	98	11.21	.20+	.35	3.36	3.51	3.87

It will be observed in Table 3 that the percentage of high myopia, 10 D. or greater, during the first period of three years, shows a steady rise in the number of such eyes from 0.80 per cent. in 1894 to 0.91 per cent. in 1895, and to 1.86 per cent. in 1896. In 1910 and 1911 not a single case of M.=10 D. was seen, reducing the average for the three years to 0.20 per cent., as compared to 1.19 per cent. for the first period. The interesting fact is set forth in Table 2 that while the percentage of the higher grades of myopia falls with more or less regularity with the succeeding years, the lower grades less than 3 D. steadily advance, not in actual numbers but in relation to the whole number of myopic eyes.

A study to ascertain if the same results would be shown by the annual reports of the staff at the Wills Hospital was disappointing. These reports, indeed, showed a slight rise in the percentage of myopia. It should be stated, however, that a constantly increasing percentage of the patients are foreign born, emigrants from southern Italy, Poland, Hungary and the Balkan States, and Russian Jews. Many of them, especially the latter, suffer from high grades of myopia and serious intra-ocular disease. They come from sweat-shops and other allied forms of employment, working with insufficient light in badly ventilated rooms, and living on insufficient and improper food, under outrageous hygienic environment. In private practice these cases are rarely seen; but even among these hospital patients the average myopia for the same period included in this report was only 15.43 per cent. The percentage of high myopia in my private records as shown in Table 2 is based on the total number of cases of myopia of 10 D. or more of only nineteen in a total of 2,297 eyes, or 0.87 per cent.

The statistics, therefore, gleaned from my private case-books show unmistakably that in my personal experience there has been a steady fall in the percentage of myopic eyes applying for treatment. This demonstrates the truth of the claim set forth in the conclusions of my report of the Philadelphia school examinations, published in April, 1881, as to the baneful influence of uncorrected errors of refraction. Furthermore, the ophthalmic surgeon has the satisfaction of seeing that his efforts in the correction of these congenital defects of refraction and faulty binocular balance have well-nigh banished from the better-to-do portion of the community the nightmare of malignant myopia and its sequelae.

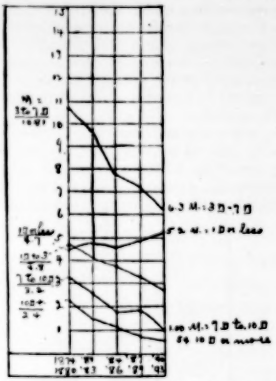


Chart 8.—Curves showing the steady fall in the percentage and degree of myopia from 1874 to 1893, inclusive, constructed from Table 1. From opticians' books.

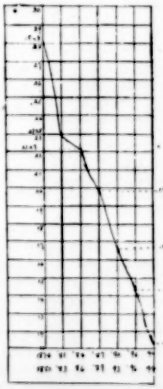


Chart 9.—Combined chart showing decrease in myopia.

2018 Chestnut Street.

ABSTRACT OF DISCUSSION

DR. F. PARK LEWIS, Buffalo, N. Y.: I do not believe that the profession has begun to realize the enormous service done by Dr. Risley in his careful investigation of refractive cases under cycloplegia, begun more than twenty-five years ago. There have been many examinations of the eyes of school-

V. 191

children before and since that time, but most of them have been under such varied conditions and with so little attention to detail that they are almost valueless for statistical purposes or for methods of comparison. Since this paper was referred to me for discussion I have again gone over the literature thoroughly and I find that the subject has been considered from two standpoints, that of the ophthalmologist and that of the teacher. To obtain a series of records made from the investigations of a careful ophthalmologist, with the accommodation of the subject placed at rest, means great labor, both in the determination of facts and in the arrangement of those facts so that they may be of subsequent use. In studying a subject for statistical purposes, let it be what it may, it is essential that the special point expected to be brought out must be particularly emphasized when the investigations are made, otherwise it is merely incidental and of comparatively little value. The purpose of this investigation, as brought out in Dr. Risley's paper, is to determine whether or not myopia is increasing. Before that question can be answered we should arrive at an understanding as to the meaning of myopia. Most ophthalmologists now recognize two definitely distinctive conditions. First, the congenital one, in which the anterior posterior axis of the eyeball is abnormally long, just as the hyperopic eye is abnormally short, taking the emmetropic eye as a standard. Those varying in length from the normal are deformed, but they are not diseased, nor are they, because of deformity, necessarily more subject to disease. On the other hand, there is an ectasia, which is definitely pathologic, in which the anterior posterior diameter lengthens with increasing rapidity, during the years of adolescence, usually ceasing to progress after the tissues of the body have become physiologically developed. I believe that these two conditions should be differentiated by distinctive names. The congenital myopic eye might continue to be so called, but the ectasia produced during the developing years should be known by some name indicating the pathology involved, such as posterior ectasia of the globe. We then would have a scientific basis which would enable us to separate the pathologic from the merely deformed eye. In the latter, so long ago as 1897, Dr. Risley said: "If heredity has any important place in the history of near-sighted eyes it lies in the production of these anatomic differences." He was of the opinion that congenital anomalies in the form of the eyeball are hereditary rather than myopia or any tendency to myopia. Dr. Risley attributed the myopic tendency to certain distortions in the form of the skull which affect the shape of the orbits. At this time the inheritance of ocular abnormalities had not been accepted. Cohn considered that the fact had not been substantiated, but the intensive study by Steiger of the eyes of 36,654 Zurich children has established Dr. Risley's contention of the inheritance of astigmatism and the relationship of astigmatism in the development of myopia, while the studies and measurements of Stilling have substantiated Dr. Risley's second contention that the form of the eyeball is largely determined by the shape of the human skull. The determination of the question as to whether or not myopia is increasing is then of more than academic interest, because if it is proved, as it seems to have been, that by abolishing astigmatism through the early adaptation of suitable correction glasses we do away with the tendency, in a large measure, to the production of myopia, we have established an etiologic fact of enormous clinical importance. This view has not yet been fully accepted. The most complete study of inheritance of defects of vision probably ever made is that by Amy Barrington and Karl Pierson which is published in the "Eugenics Laboratory Memoirs," London, 1907. In this Steiger's studies are most carefully analyzed, and among many other valuable conclusions, or rather deductions, Steiger says: "Astigmatism with the rule is always congenital; that it is never found to occur after birth, and no statistics demonstrate a more intense astigmatism according to the rule; in later years a reduction, sometimes very marked, may occur in its value." The deductions which Barrington and Pierson draw, from their studies, are that: "No sufficient or definite evidence that school environment has a deleterious effect on the eyesight of children has yet been definitely demonstrated. Undoubtedly considerable change of vision takes place during school years, marked first by a decrease in the hypermetropic classes, and an increase in the emmetropic classes. This is followed, between the ages of 10 and 14, by a decrease in the emmetropic and an increase in the hypermetropic, astigmatic and myopic classes, the balance being still in favor of emmetropia when school is left." Is the first a growth law and the second an environment natural effect? Or are both but phases of one law of growth, a passage from hypermetropia to myopia of the "unstable stocks"? This has led Professor Burnham of Clark University, in the "Encyclopedia of Education," under the heading "Myopia," to the conclusion that a certain number of children will inevitably become myopic, and, while recognizing the desirability of good school surroundings, he believes that the condition is an essentially developmental one which we are unable to modify. This brings vividly to the front Dr. Risley's contention, in which he and the rest of us, I think, are in agreement, that astigmatism is inherited; that it is on uncorrected astigmatism that myopia depends, but, and this is the essential point, that we inherit tendencies and that if astigmatism is abolished by right correcting glasses, we would thereby lessen the likelihood of the development of myopia. If this is true, and the most careful records seem to prove that it is, we have a fundamental principle of vital importance on which to proceed: The imperative necessity of the early correction of astigmatism and other forms of eye strain at that period in life when the tissues are plastic and in which they are assuming their ultimate form.

DR. ALBERT E. BULSON, JR., Fort Wayne, Ind.: Careful correction of errors of refraction in patients of all ages has done much toward the reduction of myopia, but we must also take into consideration the great advancement made in the construction of buildings, including improvements in lighting, ventilation, etc. We have also adopted the system of school examinations and otherwise put in practice many things that tend to raise the resisting power of the child, not only his general condition, but the eyes as well. Dr. Risley is so scrupulously accurate about everything that I hesitate to criticize him and yet I wish to call attention to his use of the word "mydriatic" in the place of "cycloplegic." Every cycloplegic is a mydriatic, but not every mydriatic is a cycloplegic. Euphthalmin is a mydriatic, but not a cycloplegic. When we wish to speak of paralyzing the accommodation, we ought to use the term "cycloplegia," and the agent used should be called a "cycloplegic."

DR. S. D. RISLEY, Philadelphia: If the apparent teaching of the statistics of my own work is true also for that of ophthalmologists in all parts of the United States, then the ophthalmologist has accomplished more for the conservation of vision by the correction of defects of refraction and anomalies of binocular balance than all else that has been done. When we consider the handicap for any career which the man with high myopia has; when we further consider the large numbers of people with central chorioidal atrophy, floating vitreous opacities and other sequelae of high myopia, we can hardly overestimate its value.

Necessity for Local Meat Inspection.—The monthly *Bulletin of the Indiana State Board of Health* for April prints a letter from one of the food inspectors of the State Board, who is a veterinarian. This report forcibly calls attention to the need of the inspection of meats killed outside of the large packing houses where Government inspection is provided. In the instance to which attention was called by the inspector, a cow, which had been condemned when a certain dairy was inspected, was found in a local slaughtering establishment. The animal had been milked up until the time of slaughter. The inspector found the abdominal cavity filled with miliary tubercles and also a tubercular condition of the lungs. On the protest of the inspector the meat of the animal was tanked.

POSTCATARACT EXTRACTION DELIRIUM

REPORT OF ELEVEN CASES

WALTER R. PARKER, M.D.
DETROIT

The first description of postoperative psychoses dates back to the sixteenth century when Para called attention to the occurrence of mental symptoms after operations. In 1819 Depuytren described a "delirium nervosum" which occurred in the first three days after an operation, and in 1842 psychoses after eye operations were reported by Sherzog. Sichel[1] in 1863 described a peculiar delirium which he had observed seven or eight times in the wake of cataract operations. All the patients were over 60 years old. He attributed it to sealing the lids and thought the mental effect similar to homesickness. Arlt[2] wrote in 1874:

The second eye is uncovered before the fourth day only when the patient becomes restless because of darkness. In old patients, much run down, timid and nervous, mental disturbances may occur during the first few days after the operation.

I have been unable to find a classification from the standpoint of the psychiatrist. Evidently no single form of mental disturbance is characteristic of postoperative delirium. Hurd[3] would limit the cases to those clearly the result of infective processes, while Selberg,[4] in addition to infection, includes anesthesia, drugs, auto-intoxication, physical or mental exhaustion. Picque[5] eliminates cases due to alcohol, drug intoxication and septic infections. He employs the term only to designate those instances in which the trouble supervenes in a patient not otherwise ailing. Burr[6] says that transitory confusion or delirium rarely occurs in the aged without the presence of severe arteriosclerosis and marked disease of the kidneys, even when the immediate cause is auto-intoxication from the intestines or stomach, or from some unknown endogenous source. He does not think the cerebral arterial disease is the cause of the attack, however, as practically all the aged have hardened arteries and may show no mental illness. These transitory attacks are not due to the fact that the thickened arteries do not carry enough blood to supply the brain, but are caused by a poison or poisons carried by the blood. Phillips[7] holds that in old age trivial causes may produce a wandering of the mind, due no doubt to the impaired nutrition of the nerve-cells from a diseased heart or sclerosed cerebral vessels. Gressinger, as quoted by Löwy,[8] advocates the theory that the causative factor for the origin of the psychoses lies in peripheral nerve injury or a disease of the sensory nerves originating peripherally from operative traumatism. Schultze[9] concludes that postoperative psychoses occur after all forms of operation but most frequently after "predisposed diseases." In a large number of cases a hereditary tendency is found. He agrees with Picque that definite cases of real postoperative psychoses, in contrast to the false postoperative delirium, are reduced to a small number. In the great majority of cases fever, lasting toxic condition after the operation, inanition and condition of weakness, the cause of which is to be sought in the underlying disease itself, offer the explanation for the oncoming condition of psychosis. Engelhardt[10] concludes (1) that postoperative psychoses develop in individuals predisposed by heredity, chronic intoxication, grief or care; (2) that the weakness incident to the operation or disease preceding can scarcely be considered essential—at most it may be considered as the determining factor in predisposed cases, and (3) that there are cases of postoperative psychoses in which an etiologic understanding is not as yet accessible.

It has been contended that serious operations on the pelvis and genital organs and operations on cataracts were especially prone to be followed by mental disorders. While most of the cases of postoperative delirium have been reported by ophthalmologists and gynecologists, it is generally agreed that the character of the operation itself has little or no influence in inducing a postoperative psychosis. The hereditary predisposition of the patient, his habits as to alcohol and drugs, his physical, mental and moral qualities, his attitude assumed on entering the operating-room, effect of anesthetics, dressings and complications of the operation, such as infections, all have received consideration. The effect of the darkness of the room is important, as the delirium has been observed after the application of a bandage when no operation has been performed. Schmidt-Rimpler reports two cases of delirium following confinement in the dark room without operation, one, a case of syphilitic iritis, the other case of double iridochorioiditis, in a strong young man. From these two cases, he refers the controlling visual hallucination to the shutting off of the sight sense. In cases which do not show hereditary predisposition, complications of a septic or toxic nature, disturbance of kidney or liver functions, and in which history of alcoholism and hysteria can be eliminated, the operation itself and the subsequent treatment must be reckoned as etiologic factors. Krafft-Ebing long ago suggested the possibility of fright incident to the eyes being bandaged as the exciting cause of the delirium. That the "dark cure," however, is not responsible for all cases is proved by the fact that many patients show mental disturbance when the eyes are not bandaged. Urbach,[11] for instance, reports five cases of delirium occurring in 106 operations for gall-stones. Kelly,[12] Baldy[13] and others have reported gynecologic operations which were followed by mental disturbance and in none of them were the eyes bandaged.

From this incomplete list of published cases of postoperative delirium it will be seen that the writers are not agreed as to its etiology or classification. The prognosis is generally good. But here, again, much depends on the class of cases included in the report. The age of the patient is thought to be a most important factor. A few cases have been reported in patients below the age of 60. The late onset and long duration of the mental disturbance are noticeable in these younger patients, and the prospect of recovery is not so favorable as in the old.

The cases here reported are from the ophthalmic clinic of the University of Michigan from 1908 to 1912, inclusive. The report includes cataracts of all kinds, occurring in the old and young. There are eleven cases of delirium in a total of 376 cataract extractions as shown in the accompanying table:

1. Sichel: Ann. d'ocul., 1863, xlix.
2. Arlt: Handbuch der gesamten Augenheilkunde (Graefe-Saemisch), 1874, iii, 309.
3. Hurd: Am. Jour. Obst., 1899, xxxix.
4. Selberg: Beitr. z. klin. Chir., 1904, xliv, 173.
5. Picque: Bull. et mém. Soc. de Chir., Paris, 1898, xxiv, 171.
6. Burr: Transitory Mental Confusion and Delirium in Old Age, THE JOURNAL A. M. A., Dec. 30, 1911, p. 2118.
7. Phillips: Cleveland Med. Jour., 1909, viii, 531.
8. Löwy, Rudolph: Allg. Ztschr. f. Psychiat., 1896, iii, 166.
9. Schultze: Deutsch. Ztschr. f. Chir., 1910, civ, 584.
10. Engelhardt: Deutsch. Ztschr. f. Chir., 1910, lviii, 46.
11. Urbach: Wien. klin. Wchnschr., 1907, xlvii, 1465.
12. Kelly: Surg., Gynec. and Obst., 1909, ix, 515.
13. Baldy: Am. Jour. Obst., 1897, xxiv, 1217.

DELIRIUM FOLLOWING CATARACT EXTRACTION

	Patients Operated on				Patients Delirious			
	No.	Av. Age (Yrs.).	Young-est.	Old-est.	No.	Av. Age (Yrs.).	Young-est.	Old-est.
Males	233	62.35	25	90	7	74.28	69	80
Females	143	61.05	25	86	4	68	53	82
Total	376	61.07			11	65.71		

Following is a brief history of my cases of post-cataract extraction delirium:

CASE 1.—B. M. G., woman, aged 75. Urine examination negative. April 20, 1908, simple extraction. No accident. During the evening of the second day almost maniacal, becoming uncontrollable, up and down during the night; disoriented, auditory hallucinations; tore dressings from eyes several times; wound opened, no prolapse of iris. Left hospital with daughter on third day against advice. Anterior chamber filled with blood. Returned one month later for secondary cataract operation. Eye in perfect condition. No mental disturbance after reaching home.

CASE 2.—G. W. M., man, aged 71. Urine examination negative. Oct. 22, 1908, combined extraction; no accident. Two days later delirium developed; disoriented, out of bed, fell on floor, arose and walked about the ward, calling for wife and daughter, believing himself at home. Patient put back in bed, dressings removed from unoperated eye, given ice-cap and the situation explained. He soon quieted down. No recurrence. Operation surgically perfect.

CASE 3.—G. P., man, aged 81. Urine examination not recorded. Bothered with old cystitis, passing urine involuntarily. March 1, 1909, combined extraction. Patient very restless on operating-table. Second night delirious. Bandage removed from unoperated eye. Next day patient quiet. No subsequent attacks. Eye not damaged.

CASE 4.—M. C., woman, aged 82. Urine examination negative. April 29, 1909, combined extraction. No accident. First night patient nervous and restless, sat up on edge of bed; unoperated eye uncovered. Controlled with difficulty until the third day when she became disoriented with hallucinations. Continued to have periods of disorientation until discharge on twelfth day. Eye not injured.

CASE 5.—M. T., woman, aged 62. Blood and urine negative. Dec. 12, 1909, combined extraction. Next day patient became delirious, fairly well oriented but suspicious of those about her, garrulous, and not amenable to discipline. This continued until patient was discharged on the twelfth day. Operation surgically perfect. No further record.

CASE 6.—B. S., man, aged 77. Urine negative, Aug. 15, 1910, combined extraction, given codein 1 grain. During evening of second day patient out of bed, talked incoherently, disoriented; given morphin sulphate ¼ grain hypodermically, with little effect. Patient restless all night, out of bed several times. Following day thought he was in a barn, but did not know doctor or nurse, refused to go back to bed. Anterior chamber filled with blood. Given morphin ¼ grain. Slept during part of the day. Fifth day increased restlessness all afternoon. He knew he was in a hospital but thought his son was in a near-by bed, and had been subjected to an operation. This condition continued until the tenth day when he became oriented and quiet. Result of operation perfect.

CASE 7.—F. K., man, aged 80. Urine normal. May 1, 1911, combined extraction. On following day became delirious. Out of bed twice, tore pads from both eyes. Played with loops and picked at bed-clothes. Given veronal without effect. Up and walking about during the night. Could not be induced to go to bed; highly excited, talked incoherently. Violently resisted any attempt to put him back to bed. Tried to escape from room by pushing doctor and nurse aside. Repeatedly tore pads from eyes. Given scopolamin 1/1000 grain. After half hour slept for six hours. Constantly confused and disoriented until discharged on eighteenth day. Eye not injured. No subsequent record obtained.

CASE 8.—M. C., man, aged 70. Urine examination negative. Oct. 15, 1911, combined extraction. At midnight of second day after operation he became confused, tore bandages from his eyes and wandered about the room. Was quieted by intern, became oriented, gave as excuse he had wakened suddenly and could not locate himself. Thereafter convalescence was uninterrupted. Eye not injured.

CASE 9.—M. S., female, aged 53. No record of urine examination. Jan. 25, 1912, combined extraction. No accident; given codein 1 grain. Considerable reaction followed the operation. Patient was nervous and apprehensive but suffered no delirium until sixth day when she became disoriented and talked irrationally. Refused to eat, claiming food contained cut glass. Several times attempted to get out of the room and to go to see the doctor. Thought she heard some one calling her name. On the ninth day more quiet, but constantly watchful, frequently asserting she might be harmed. Never again disoriented. Always knew the names of those about her. Auditory hallucinations continued until the time of discharge on the twenty-first day.

CASE 10.—M. E., man, aged 72. Urine examination negative. July 17, 1912, combined extraction without accident. During night of second day after operation patient became delirious; nurse unable to control him. Out of bed, disoriented, had hallucinations of hearing and talked in a rambling manner. Pad was removed from unopened eye. Later he became nauseated and vomited a small amount of undigested food. No signs of delirium followed. Eye not injured.

CASE 11.—M. S., man, aged 69. Urine negative. July 12, 1912, combined extraction. During afternoon of second day became delirious. During night much worse, with hallucinations of hearing and disorientation. Given morphin sulphate ¼ grain and tepid sponge bath; removed pad from unoperated eye. Later given 1/1000 grain scopolamin, after which he slept three hours. On waking the delirium was still marked. Next day he was quiet and had no return of delirium. Eye not injured.

It will be noted, in the cases here reported, that the psychoses occurred only in the aged, the youngest male affected being 69 years old, the oldest 80, average 74.28; the youngest female 53, the oldest 82, average 68 years of age. Males were more frequently affected than females, seven patients being men and four women.

The psychoses began from twenty-four hours to six days after the bandages were applied. In five cases the delirium occurred during the daytime, in six during the night, but in every case the symptoms were more prominent at night.

In no case did the patient show signs of deviation from a normal mental state, either before or at the time of operation. A general anesthetic was not administered. Four instillations of 4 per cent. solution of cocain were used at intervals of two minutes in each case. After operation the patients were quiet and tractable. Later they became restless, indifferent to precautions to remain quiet, often raised themselves without assistance, disturbed the bandages, etc. They usually answered questions rationally, but gave little heed to instructions. With some patients the first manifestations become more marked at night; they get out of bed, perhaps allowed themselves to be quieted for a short time, or became obstinate, scolded and defended themselves. The majority were disoriented and hallucinations of hearing were more common that those of sight.

One of the most remarkable things about the series of cases here reported is that in not a single instance was the eye permanently damaged by the conduct of the patient during the delirium. Seven of the eleven cases became rational, and were apparently well before leaving the hospital. One left on the third day still unbalanced, but returned in one month, with a history of having had no mental disturbance after reaching home. Three were still delirious when they left the hospital,

one on the twelfth, one on the eighteenth and one on the twenty-first day. No subsequent history was obtained in any one of these cases. Fever was not present in any case.

As a careful history of hereditary psychosis was not included in my notes and but two of the cases were studied by a psychiatrist, the mental disturbances cannot be classified. The following observations, however, may be made:

1. The delirium occurred in 0.29 per cent. of the cases operated.

2. No patient showed marked signs of mental disturbance while under observation, either before or at the time of operation.

3. One case showed possibility of infection from an old cystitis.

4. The urine was free from sugar, albumin or casts in nine cases. No record was made in two cases.

5. Codein was administered in two cases, 1 grain hypodermically, immediately after the operation.

6. The possible effects of cocain can be eliminated, as the mental disturbances did not occur in a single case until at least twenty-four hours after the time of operation, and there was no rise in temperature.

32 Adams Avenue West.

ABSTRACT OF DISCUSSION

DR. EDWARD JACKSON, Denver: I recall one case in which postoperative delirium was followed by a serious result. A patient at Wills Hospital some years ago, in his delirium got out of a second-story window, fell and broke his femur. I have never seen a serious result to the eye. Possibly, without showing evidence of it, the patient has the eye very much in mind and so avoids doing things that would involve it. It is quite possible that consciousness of something important done to the eye is an element in the delirium. Several years ago I saw mydriatic delirium in an elderly patient. It is possible for mydriatic delirium to arise from moderate doses of atropin, if continued through several days. It is well recognized that the wearing of a belladonna plaster may, after many days, produce delirium. We commonly think of such delirium as occurring in children but I have seen it several times in elderly people. In these cases of Dr. Parker's it can be ruled out as can other forms of poisoning or the results of septic conditions. We have, therefore, the pure postoperative delirium. Perhaps it is more noted in connection with cataract operations because there is less general disturbance of the patient. I am inclined to think that it is due largely to the element which Dr. Parker brings out in his statistics on the age of the patient: that it is a disturbance induced in a person who is undergoing senile changes affecting the mental processes. The disturbances of the patient do not seem serious to us and yet they are more so than we are inclined to estimate. These patients undergo a complete change of life and routine at a time when they are not mentally able to withstand the effect of the shock under circumstances of severe excitement. I think that the bandaging of the good eye, when it retains some sight, and perhaps the darkness of the room and the quiet, producing a different set of impressions through the sensory channels that still remain open, constitute another causative element. It seems to me it is distinctly a psychosis brought about by mental influences, best guarded against by reducing the changes from the normal life of the individual to the minimum.

DR. ALLEN GREENWOOD, Boston: This condition might be called a form of mental shock, and as Dr. Jackson has suggested, will occur almost invariably among hospital patients. I have had two severe cases in the hospital. The first was a woman, aged 77, who at the time of her death was still insane. She never recovered her mental equilibrium after the postoperative delirium. The other patient was delirious for about forty-eight hours. The point in Dr. Parker's paper that is of particular value at this time is the necessity of accustoming the patients in some way to their hospital surroundings, and since seeing one patient permanently insane I have been trying to follow out this plan. In my private practice, if the patient lives near me, I am in the habit of operating in the home. With reference to this particular point I believe that I get better results, with good aseptic precautions and a trained nurse and with the patients in their home surroundings, among friends, than I can get in the hospitals.

DR. C. D. WESCOTT, Chicago: I have had one fatality following the delirium Dr. Parker has described. There was nothing unusual about the operation. The delirium began within forty-eight hours after a perfectly smooth extraction in a patient aged 78, with marked arteriosclerosis. The bandages were removed and the wound opened, but we succeeded in controlling him with sedatives and the constant attendance of nurses. The wound closed and on the tenth day he seemed to be perfectly well and was happy at the prospect of going home. That day the special nurse was taken off his case. The night nurse saw him at 12 o'clock and all was well. At 12:15 she heard a crash. The patient had gotten out of bed, run out of the room and jumped over the rail surrounding the staircase. He was found helpless at the foot of the stairs and died in ten minutes. This may have been the result of a bad dream or an attack of senile delirium. My father had an attack of senile delirium on a train. It was a hot day and he was much fatigued. During the first day or two of the attack if his eyes had been bandaged he would certainly have taken the bandages off. The delirium did not differ in any respect from that described by Dr. Parker. I believe the senile aspect must be considered and it is possible that my patient had recovered from the delirium following the operation and that the disturbance in which the fatality occurred was a secondary affair.

DR. MELVILLE BLACK, Denver: It seems to me the thing to be especially considered in these cases is to afford absolute mental rest. This can be accomplished by many operators through suggestion. Not all of us, unfortunately, are given the same power in that direction. I have endeavored to give my cataract patients absolute mental and physical rest by the administration of ¼ grain of morphin a half-hour before the extraction, repeating the dose six hours later. This makes the patients quiet even if they are not asleep. Their minds are at rest and they are not worrying over the possibilities of the outcome. I think we are absolutely justified in saying to the patient that the operation is a simple one and that there can be absolutely no doubt of a perfect result. It would be proper, as a matter of precaution, to explain to the other members of the family the risks to be taken, but the patient should be given the idea that there is no possibility of failure. I think this has a great deal to do with the outcome in these cases. Another point is to get these patients out of bed. I usually operate in the afternoon between 3 and 5 o'clock while the light is still good, and the next morning when I see the patients I ask them if they want to get up. They invariably say yes, and I have no objection to their sitting up. The recumbent position is often restful to many people, but not so to others. The idea is to give them rest and I believe this is not only permissible but advisable. There are a great many factors to be considered from the standpoint of the individual patient.

DR. WILLIAM ZENTMAYER, Philadelphia: I want to cite a case showing that it is not always the removal of patients from their homes which is responsible for the delirium. This case occurred early in my practice. An elderly man was operated on in his home. On the second day after the operation, because of the illness of the surgeon, I was called to see the patient, who was in violent delirium. He recovered under scopolamin and croton oil, which I believe I was foolish enough to give him. A second case had so many contributing factors that the direct cause of the delirium is obscure. An elderly woman was brought from a distant city for extraction of a cataract from an extremely myopic eye. She had been practically blind for three years. The immediate operation was

V. 6
1913

successful, but she was in the hospital for three weeks with a low-grade iritis. She was then sent to the suburbs against her wish. She knew that her home in which she had lived for many years was to be closed during her absence. The day before the attack her son came to visit her. She also knew I was going away. The night before I left she was taken with a delirium which lasted throughout the night. I saw her early the next morning and found her somewhat improved but I have not learned the outcome.

DR. S. D. RISLEY, Philadelphia: I have seen many cases of postoperative delirium and while the majority of the patients have recovered I have seen enough cases prove serious to make me anxious about them. For example, one man left his bed, jumped from the second-story window and was killed. I am sure that being left in the dark is not always the cause of the delirium but I have seen the delirium disappear after removal of the bandage from the unoperated eye. I was interested in the remark made by Dr. Black about the administration of morphin. I do not as a rule approve of the administration of morphin after the extraction operation, or any other, because it is apt to conceal symptoms and disturb the general nutrition of the patient. In most cases of postoperative delirium I believe it is theoretically correct to administer a cerebral stimulant. The morphin or some preparation of opium should be administered, if at all, in small doses, but instead of the opium I prefer hyoscyamin or stramonium in small doses. In some cases, however, I have found more prompt relief from stimulating tonics. A routine mixture of mine for old people after operation is the tincture of nux vomica, compound tincture of cinchona and compound gentian. In two instances I have seen the delirium disappear after the man was taken out of the room and given his pipe. Patients have asked if they could smoke and I have told them they could have a dry smoke. I get old people out of bed as soon as possible. It is a serious thing to put a man 75 years old in bed away from his usual environment and activities.

DR. WALTER R. PARKER, Detroit: In practically all that has been written on the subject the argument is made that senility is not the only etiologic factor in postoperative psychoses or the delirium would be much more frequent. A predisposition to some psychoses, associated with senility, is generally the determining factor. My experience agrees entirely with that of Dr. Risley in regard to the treatment of these cases. In every case the unoperated eye has been unbandaged as soon as the delirium was discovered and usually the patient improved mentally. Scopolamin, morphin and bromids have been used and in some cases appeared to have good effect. One patient, however, was made much more delirious for a short period by a dose of scopolamin. I cannot name any one drug as being of special benefit in routine treatment. I believe we do not give enough attention to the general welfare of these cataract patients. After the operation I put the patients immediately in a small ward with two beds and a special nurse. They remain there until the anterior chamber is closed and then they are put in the general ward. Two cases developed a delirium immediately on being placed in the general ward. If these patients could have remained in a private ward under the care of a nurse it is possible the delirium would not have developed.

Duodenal Ulcer.—In the early days of our recognition of this condition we were assailed by a formidable array of post-mortem statistics which went to show the rarity of the disorder, and the absence or the inconstancy of any symptoms which could be attributed to it during the life of the patient. We now know that the great frequency of the disease—every month I operate in more cases than were formerly recognized in the post-mortem room of the largest hospitals in ten years—and the assertion may truthfully be made that of all abdominal diseases none can be more certainly discovered by a scrutiny of the anamnesis alone.—Sir Berkeley Moynihan, Address in Surgery, Brit. Med. Assn., 1913.

THE SURGICAL TREATMENT OF A CERTAIN TYPE OF PENETRATING WOUNDS OF THE SCLERA, BY MEANS OF A DOUBLE CONJUNCTIVAL FLAP

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The use of some form of conjunctival plasty to close and reinforce penetrating wounds of the sclera is a too well-established principle of surgery to need elaboration, although, when Kuhnt published his work, as great a surgeon as Herman Knapp expressed himself as being somewhat dubious as to whether Kuhnt had materially improved on Nature's way. To Kuhnt, of course, belongs credit for having been one of the foremost and most persistent advocates of the use of the conjunctiva in ocular wounds. Nothing of an essentially fundamental nature can be added to the surgical principle as has been set forth by him.

It is the purpose of this paper to discuss the surgical treatment of a certain type of penetrating wounds, in reference to the advisability of the use of scleral sutures, and to call attention to a method of sealing the wound by means of a double conjunctival flap in such a way as to avoid their use if possible. After a review of the current books on ocular surgery, it may be said that most authors divide penetrating scleral wounds into two groups: those not requiring scleral sutures and those that do; such a division being based on the liability of wounds to gape. In the first class are placed wounds of less than 3 mm. and into the second fall those of 3 mm. or more. Such a division, of course, is not to be taken literally, as the individual case may warrant modification. In the main, however, it expresses the sentiment of many conservative surgeons. Something more than mere size must naturally enter into such a consideration; especially is this true as to the location of the wound in regard to tension from the neighboring ocular muscles; a wound situated so as to be exposed to the direct pull of internal or external rectus is more apt to gape than one further removed.

The end sought by all surgical procedures is the rapid and firm coaptation of the wound lips, and formation of as thick and unyielding cicatrix as is possible, looking forward to the avoidance of subsequent cystoid bulging. It has been shown that the healing process in scleral wounds is not one of direct adhesion but of a deposition of new connective tissue. The source of this new scar tissue is not the sclera, according to Lublinsky, but the conjunctiva and chorioid. If these observations are correct the sclera itself plays quite a passive rôle, providing merely a basis on which the scar is built up by cells which are of conjunctival or chorioidal origin. As far as the sclera is concerned the surgical problem is entirely one of drawing and holding the wound edges together, so as to permit the conjunctival and scleral tissues to do their repair work best. While this fact alone does not present a powerful argument against the scleral suture, it deserves consideration. Of still more moment is the almost necessarily severe manipulation which scleral stitching involves. Scleral tissue is too tough and resisting to admit of ready suturing. Along with the added surgical trauma goes the danger from loss of vitreous. If the conjunctiva can be manipulated in such a way as to exert enough traction on the wound margins to bring and hold them together, and at the same time to provide an ample reinforce-

ment, then the more difficult scleral suture may be avoided, and with the added insurance of a firm scar.

The type of penetrating scleral wounds, to which the following procedure is applicable, must of necessity be those located far behind the corneoscleral junction, to admit of relatively free elevation on all sides of the neighboring conjunctiva. It is further assumed that such wounds fall under the group of those which, according to sound surgical principles, are favorable for closure. It includes, however, those of any shape or extent, whether ragged or smooth. First of all, tags should be trimmed down, presenting vitreous clipped, and the wound edges made ready for coaptation. It is in this step of the procedure that I think I have an improved technic. At any rate, I have not seen this method described in anything I have at hand. By means of strabismus or tenotomy hook, the conjunctiva on all sides of the lip of the wound is freely loosened from the sclera. In a radial wound, it is my practice so to free the conjunctiva on each side, for about a quarter of the way around the eyeball and to a relative extent in the other directions; if equatorial, as far forward as the corneoscleral junction. Double-armed fine silk sutures, one or two, depending on the size of the cut to be closed, are then placed in the margin of one of the newly made conjunctival flaps. The needle or needles, as the case may be, are then carried well up under the other flap, brought to the surface and firmly knotted. The result is to tuck up one flap under the other and bring one layer of intact conjunctiva over the scleral wound. The

The dotted line shows the outline of the under flap which is tucked up under the upper flap. The lines of traction on the two flaps are so arranged that the wound lips are held in firm apposition.

surface of this flap is then carefully abraded, usually by gentle scraping with a knife. The remaining conjunctival flap is then brought over the first and stitched down in the opposite direction. When completed the wound is closed by the second layer of healthy conjunctiva and correspondingly reinforced. In drawing over the flaps, in the manner just indicated, it is important that the sutures be snugly drawn and firmly knotted. Otherwise one of the most important features of the operation will be lost, namely, traction on the globe in such a way as to bring together the scleral edges and hold them firmly in place.

If, however, the wound is of such size and location that the operator does not care to trust entirely the conjunctival flaps, and deems it wise to close the sclera as well, this should be done before the conjunctival flaps are freed. The surgeon has the choice of several suture materials. As these sutures are to be buried, they must be absorbable. Very fine catgut, kangaroo and rat-tail tendon, are to be had. Of these the latter appears best adapted to the work in hand. It is strong and yet extremely pliable. This latter property minimizes the size of the suture canal. Whatever material is chosen, lubrication with sterile olive oil or petrolatum will facilitate stitching and also give the added advantage of filling the suture canal with an aseptic material. If not of greater, certainly of equal importance as the choice of suture material is the absolute necessity of sharp, fine needles. A dull-pointed, thick needle causes immeasurable and irreparable damage, while a sharp, fine needle not only facilitates the placing of stitches but reduces danger from added traumatism. Whether the stitches should include the entire sclera or whether they should be only episcleral is a question which I do not pretend to decide any further than to say that in my own experience, I have ever found the latter satisfactory, both in immediate results and in cases seen seven years after operation.

The above-described technic is available whether or not scleral sutures are thought necessary. I have not used the scleral sutures since this procedure was adopted, although a fair amount of these kinds of accidents have been dealt with at the Emergency Hospital and other hospital services and in private practice. Since the double flap method, as described, has been employed, I am convinced, in my own mind at least, that the results have been more satisfactory, healing more prompt and kindly than when the single flap or plain conjunctival sutures were employed. Such a statement is, however, open to the just criticism that there is no ground for a fair comparison.

The following points are mentioned in favor of double flap as compared with the single:

1. Because of the traction exerted by the two flaps, the scleral wound lips are held in firm apposition. Consequently, relatively large scleral wounds may be rapidly and safely closed without stitching the sclera.

2. The resulting scar is thicker, firmer and more unyielding.

3. Two layers of sound conjunctiva protect the contents of the globe from outside infection.

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ABSTRACT OF DISCUSSION

DR. JOHN A. DONOVAN, Butte, Mont.: This operation is well worth a fair trial. It may be that the additional support of the double flap will prove of decided advantage where closure is indicated, as of course it is in many cases. There seems to be no specific treatment applicable to any two particular injuries. The question comes up as to the necessity of closing the wound at all, or whether by just letting the two edges come together we would not get a better circulation. Many of the patients I see are injured in the shops and on the railroad many miles away, so that it is a day or two before they get in and in many instances the wounds are already closed. If there is no foreign body in the eyes I invariably leave them closed. The results of leaving them alone are better than interfering. I have found in practically all injuries the less you do the better off you are. The greatest danger of infection is at the time of the operation. When I see a bad wound in the sclera, with the vitreous protruding, I have been inclined to get the eyelid closed as quickly as possible, and unless I see decided indications to the contrary, notwithstanding the authorities who say that they should be covered, my results have been quite good from cleansing the wound, closing the eyeball, making gentle pressure and leaving it alone.

DR. W. H. WILDER, Chicago: The question of whether or not it is best to suture scleral wounds is, of course, an important one. I have found that there are a number of cases in which this could be done satisfactorily without putting in an absorbable suture. I have made use of a little procedure which I have not seen described and which has proved satisfactory in the few cases in which I have used it. It is applicable to any wound in the sclera. The method must be adapted to the wound; in other words, we cannot have any-

thing like a typical operation in these cases. The conjunctiva is loosened for a considerable distance around the scleral wound, which is then carefully freed of any prolapsed chorioid or ciliary body. A fine silk suture, doubly armed with fine needles, is then passed through the lips of the scleral wound, and both needles of the suture are passed through the conjunctiva near each other at a point some distance from the edge of the wound in the conjunctiva. If the conjunctival flap is now drawn over the scleral wound, the suture, when tied, will close the wound in the sclera and yet be on the outside of the conjunctiva so that it can be readily removed when desired. The flap of conjunctiva can then be secured under the opposite loosened conjunctiva by sutures similar to those used in transplanting a pterygium by the McReynolds method.

DR. ALLEN GREENWOOD, Boston: There are two or three things to be remembered. An eye, usually from which some vitreous has escaped, must be handled with great care, making this one of the most delicate operations in ophthalmic surgery. If you do not sew up a wound in the sclera in an eye that has a protrusion of vitreous and ciliary body, the eye is pretty sure to be lost, and I know of some eyes that were saved by what I would call conservative surgery. In one case in which the fundus of the eye could be seen through the wound in the sclera I did an advancement of the conjunctiva underneath the upper layer, being careful that neither my assistant nor I made any pressure on the eyeball. The man has good vision to-day and I feel that if it had been left open I never would have secured so good a result. These operations apply particularly to the cases a short time after the injury. Dr. Donovan sees his cases much later, which gives us a different problem. I think Dr. Francis has given us a valuable suggestion.

DR. LEE MASTEN FRANCIS, Buffalo: The point of this procedure is to avoid stitching the sclera, which perhaps in the experience of an operator such as Dr. Wilder is not important, but it is something which I approach with a great deal of respect. I believe that a relatively large scleral wound can be closed with the double conjunctival flap, and the strong point about the procedure is that the flap not only covers the wound but it exercises traction on the wound along proper lines.

PRIMARY LUES OF THE BULBAR CONJUNCTIVA

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Statistics show that six or seven out of every hundred cases of chancre are extragenital. Fournier[1] says, "I think the proportion of 9 or 10 per cent. would not be too high for extragenital chancres." Although they have been noted on all parts of the body, literally from head to toe, the ophthalmologist rarely meets this condition. Thus Zeissl, in 4,000 cases of syphilis, observed only two chancres on the lids, while Boeck of Christiania had one patient with palpebral chancre in 2,344 cases. In 642 cases of extragenital chancre, Fournier observed only seven on the eyes or lids (location not stated). Bulkley[2] personally observed 113 cases of extragenital chancre, four of which were on the eyelids. He has tabulated 9,058 cases, 372 of which were of the lids or conjunctiva.

In relative frequency, the structures of the eye which may be involved, are, according to Ronvillois[3]: the ciliary margin, skin of the lid, palpebral conjunctiva, culdesac, bulbar conjunctiva (scleral portion, inner angle and caruncle), limbus and cornea. The location of extragenital chancres about the eyes in the ninety-four cases collected by deBeck[4] was as follows: thirty-five on the lower lid margin, inner surface and culdesac; twenty-five on the inner angle and caruncle; twenty-four on the upper lid and culdesac; six on the ocular conjunctiva; four on the skin. The frequent involvement of the commissures and lid margins is due to the maceration of the epithelium by tears, and the resulting ulcerations, excoriations and blepharitis, offering opportunity for infection. The caruncle, plica semilunaris and lid margins are frequently irritated by scratching with the fingers.

Up to the present I have succeeded in finding but twenty-one cases of chancre of the bulbar conjunctiva, and of these only three were at the limbus. The report of a case of chancre of the limbus follows:

M. W., aged 37, an unmarried woman, was referred to me, June 18, 1909. She began the care of her sister's 3-weeks-old infant, March 25, 1909. The child showed evidences of hereditary lues. It weighed less than 6 pounds, was prematurely born, and had the appearance of an old man; the head was large and the skin was wrinkled. Its cry was weak, and it had spots on the face and body, blisters about the anus and between the fingers, a discharge from the nose, and bleeding from the mouth. The child died when it was 4 months old.

Present Illness.—May 25 the patient had a sudden pain in the left eye. She thinks that the eye may have been inflamed several days, as the upper lid covered the part of the globe most involved. The eye became very red, there was considerable lacrimation and the lids were moderately swollen. The pain was so severe that for several nights she walked the floor, being unable to sleep. The pain stopped rather suddenly two days previous to my examination, June 18, 1909.

Primary lues of the bulbar conjunctiva.

Examination.—V. O. D.=6/5. V. O. S.=3/50. The lids were entirely closed, owing to swelling. There was marked chemosis of the bulbar conjunctiva, of a deep dusky red, which extended above to the culdesac. On the bulbar conjunctiva, in the superior temporal quadrant, at the limbus, was a hard crescent-shaped area 1.4 cm. in diameter. This extended on the temporal side to below the level of the pupil, and was raised about 2 mm. above the level of the cornea. The center was occupied by a dirty, grayish-yellow necrotic area, with deep, sloping, regular edges. The ulcer was oval in outline, measuring 4 by 6 mm. The long axis was horizontal. The lower edge of the ulcer extended to the cornea. The latter was not ulcerated, but showed a deep infiltration in the portion near the ulcer. The pupil was dilated with atropin. The fundus was not distinctly seen on account of haze of the cornea. The left preauricular lymph-node was hard but not tender, and measured about 1.5 cm. in diameter. There was also some enlargement of the submaxillary lymph-nodes.

Diagnosis of chancre was made, and the patient put on inunction of mercurial ointment. Cultures showed staphylococci, and *Spirochaeta pallida* was found in the scrapings from the ulcer.

Subsequent History.—July 20 the ulcer was healing, the chemosis was less, the conjunctiva was becoming pale, but slight induration was still present, and the preauricular lymph-node was enlarged. The inunctions were discontinued and mercury, 2/5 grain three times a day in the form of an enteric pill,

1. Fournier, J. A.: Treatment and Prophylaxis of Syphilis, Am. ed.
2. Bulkley: Syphilis of the Innocent, New York, 1894.
3. Ronvillois: Rev. gén. d'opht., July, 1909.
4. DeBeck: Hard Chancre of the Eyelids and Conjunctiva, Cincinnati, 1886; Tr. Am. Ophthal. Soc., July, 1886.

was ordered. Sept. 1, 1909, the eye was white and there was a white scar on the upper part of the cornea. V. O. S. = — 0.50, + 2.00 cyl. axis 90° = 6/9 +. The patient was seen again November 23, with an iritis in the left eye. This along with a papular rash commenced two weeks previously. Inunctions were ordered again, as the patient had neglected her treatment for over a month. December 6, an iritis appeared in the right eye. The attack of iritis in the second eye was of short duration. Both eyes were white, December 22, Sept. 20, 1910, V. O. S. = 6/12 with glass.

ANALYSIS OF CASES

In a review of the literature quite a large number of cases of chancre of the caruncle and culdesac were noted. these are, of course, not included in this paper. Of the twenty-two cases of chancre situated on the bulbar conjunctiva, seventeen are reported in French literature.

Eye Involved.—The lesion was situated in ten cases on the right eye, in six on the left, and in six cases the location was not given.

Age.—All these cases, except one, have been in adults. This is rather remarkable when one considers how much infants and children are kissed and fondled. Ages reported by different writers are as follows: 6 years, DeWecker;[5] 18 years, Williams;[6] 22 years, Sans Blanco;[7] 23 years, DeWecker;[5] 24 years, Ronvillois,[3] Baudon;[8] 25 years, Valude and Laur;[9] 26 years, Horand,[10] Clergerie;[11] 27 years, Morax,[12] Fanc,[13] Maslenikow;[14] 30 years, Pelissier,[15] DeWecker;[5] 35 years, Sourdille,[16] DeSchweinitz,[17] Sbordone;[18] 37 years, my case.

Sex.—Nine males and thirteen females were reported.

Occupation.—Two married women, 5 nurse-maids, 3 laborers, 2 physicians, 1 football player, 9 not stated. The number of nurse-maids shows the danger of infection from syphilitic infants.

Situation of Chancre.—Eight were on the nasal position of bulbus; 2 on the inferior nasal quadrant; 1 on the inferior; 1 on inferior temporal side; 4 on the temporal side; 4 at the limbus (1 each on the nasal, inferior and superior portions), 2 not stated.

Mode of Infection.—1. Direct: By kissing or coughing, the saliva from a person with mucous patches may infect another, as in the cases of Hogge,[19] DeWecker[5] (two cases), Sourdville,[16] and Sans Blanco.[7] Fournier[1] treated five cases of chancre of the eye (situation not given) among his medical colleagues, the infection being transmitted in each case by the patient coughing or spitting, while naving the throat examined. Three of these washed their faces immediately after the occurrence. As he says, this does not show the futility of washing, but rather the necessity in such cases of complete ablutions with antiseptics, such as mercuric chlorid (1:1,000).

DeSchweinitz[17] reports the case of a physician who developed a chancre of the bulbar conjunctiva as a result of some secretion entering his eye, during attendance on a case of parturition. The practice among some people of using the tongue to remove foreign bodies from the eye may prove a source of infection. Fournier[1] mentions an account of an epidemic of syphilis described by a Russian physician, Tepljaschin, which was introduced into two villages by a "magician," who professed to cure trachoma and to remove foreign bodies from the lids, with the tongue. Sixty-eight persons were contaminated; eight of the first victims developed chancres of the eyelids. DeWecker's[5] third case and probably Horand's[10], are examples of this.

2. Indirect: The infection may be carried to the eye indirectly by the fingers, as in the cases of Ronvillois and Sbordone. The cases reported by Williams,[6] Baudon,[8] Dagneloiski,[20] Morax,[12] Clergerie[11] and by me likewise come in this class, a luetic child in each case being the source of the infection, and its caretaker the one infected.

3. Mediate: Infected surgical instruments, towels, handkerchiefs and sponges may spread the contagion. Such is, however, probably unusual and difficult to trace.

Period of Incubation.—This is difficult to determine, as generally the time and mode of infection are not known. In DeSchweinitz'[17] case the lesion developed six weeks after the infection took place. Ronvillois'[3] case developed less than one week after the supposed infection, a scratch from a finger. The short time of incubation in this case is against the scratch as the cause of the infection. Ten days is the shortest time given for the period of incubation of a chancre.

SYMPTOMS

Onset.—This generally resembles a mild conjunctival irritation, with localized redness. There is slight lacrimation but no secretion. Pain has been noted in only those cases in which the chancre was situated at the limbus. The redness changes to a papule-like area, the center of which becomes necrotic, forming an ulcer.

Stage of the Developed Chancre.—Ten days to two weeks after onset, the condition is characterized by chemosis; an ulcerated area surrounded by a red, raised, indurated ring, and an enlargement of the preauricular, and frequently the submaxillary gland. The chemosis is marked, involving the bulbar conjunctiva and extending into the culdesac. It is red, gelatinous-like, and has the appearance of subconjunctival injection of oil. The color has been described as wine-red, yellow-red and copper-red. It appears at about the same time as the ulceration, and continues some time after the latter is healed.

Ulceration.—This is probably present in every case. It is generally single, but may be multiple. Vignes',[21] Sourdille's[16] and Ronvillois'[3] patients each had two ulcers; Dagneloiski's[20] three. These multiple ulcers may unite and form one large area. The size of the ulcer is generally about 4 by 6 mm., and is round or oval in outline. It is of a dirty grayish-yellow, and has been described as diphtheritic or mucous-like, with an opalescent appearance. The exudate is of a mucoid consistency. The edges of the ulcer are fairly high, sloping and regular in outline. The chancre is typical, a raised dusky red, indurated area, of the consistency of cartilage, not tender or painful. In appearance it is like a button with a punched-out yellow center. In size it averages about 15 mm. in diameter.

Corneal Involvment.—Cloudiness of the cornea was present in the case reported by Fano.[13] DeSchweinitz's[17]

5. DeWecker: Cited by deBeck (Footnote 4).
6. Williams: Liverpool Med.-Chir. Jour., 1884, iv, 195.
7. Sans Blanco: Arch. d. Oftal. Hispano-Americanos, T. I. 1901, p. 52.
8. Baudon: Rec. d'ophtal., 1885, vii, 673.
9. Valude and Laur: Ann. de dermat. et de syph., 1897, Part 3, p. 294.
10. Horand: Traité pratique des maladies vénériennes, Jullian, 1886, p. 587.
11. Clergerie: Arch. d'ophtal., 1910, xxx, 43.
12. Morax: Ann. d'ocul., 1900, p. 297.
13. Fano: Jour. d'ocul. et de Chir., Nov. 25, 1874.
14. Maslenikow: Arch. d'ophtal., 1902, p. 540.
15. Pelissier: These, Lyon, 1904.
16. Sourdille: Gaz. méd. de Nantes, 1900, xviii, 113.
17. DeSchweinitz: Ophthal. Rec., 1898, vii, 144.
18. Sbordone: Movimento, Napoli, 1882, xiv, 155.
19. Hogge: Ann. Soc. de med. de Liege, 1894, No. 33, p. 94.
20. Dagneloiski: Ann. d'ocul., 1895, cited by Pelissier (Footnote 15).
21. Vignes: Soc. d'ophtal., Paris, Nov. 7, 1893; Ann. d'ocul., 1893, cx, 358.

case is unique in the development of a vascular keratitis. This subsided after the disappearance of the lesion of the bulbar conjunctiva, leaving a slight haze to the cornea.

Pain.—This has been noted only in those cases in which the lesion was located at the limbus (Fano,[13] Maslenikow,[14] Sbordone,[15] myself).

Period of Healing.—The ulceration gradually becomes smaller; at the same time the chemosis disappears, and the conjunctiva blanches. The induration persists some time after the chemosis and ulceration. The ulcer is finally replaced by a white scar, or, as described by others, by a copper-colored area. The course of the disease is generally from six to ten weeks.

Adenopathy.—The preauricular lymph-node on the same side is enlarged, hard, but not tender, and does not break down. In one case, Ronvillois',[9] the preauricular lymph-nodes of both sides were enlarged. In about half the cases the submaxillary lymph-nodes were enlarged. The adenopathy is contemporaneous with the ulceration and the chemosis. The secretion is scanty; lacrimation is present.

Pathology.—Only one case (Sourdille's[16]), has been examined microscopically. Sections were made so as to include the entire thickness of the lesion. The picture was characteristic of chancre. The infiltration of lymphoid cells was especially marked about the numerous blood-vessels, forming a kind of perivascular "cuff." Multinucleated epithelioid cells and leukocytes were also present. The endothelium appeared normal, but the tunica externa was thickened and many vessels contained thrombi. This infiltration gave rise to the cartilage-like hardness. The epithelium over the ulcer had disappeared, and the base consisted of a necrotic mass. The loose subconjunctival tissue was infiltrated by a serous coagulum in which were a few round cells. The thrombosis of the vessels was the cause of the ulceration and chemosis.

DIAGNOSIS

In the early stage the diagnosis is difficult. The condition might easily be mistaken for conjunctivitis, episcleritis or a phlyctenule. At the end of two weeks, when the chancre is developed, diagnosis should be certain, if the condition is thought of. Marked chemosis, ulceration, induration and enlargement of preauricular lymph-nodes are constant. To this may be added lacrimation and absence of pain, unless the chancre is situated at the limbus. The diagnosis is made absolute by the demonstration of the *Spirochaeta pallida*. The Wassermann reaction is not positive before the third or fourth week. The following several conditions might be confused with chancre:

Chancroid.—The induration about the ulcer is absent. The edges are sharp-cut, there is more or less itching, and there is an abundant secretion, in which the bacillus of Unna-Ducrey is found.

Tuberculosis.—The disease appears in several clinical forms. The lesions may consist of several rounded areas, similar to phlyctenules, or these may be multiple small ulcers, from 3 to 5 mm. from the limbus. The secretion is scanty, although in the large ulcers with rough, overhanging, granular, indolent edges, the secretion may be profuse. In these cases there is no chemosis, and the hard infiltration of the ulcer margins is absent. The pain is often severe. The disease is chronic and of long duration. The tuberculin test, the demonstration of tubercle bacilli or animal inoculations would determine the diagnosis.

Scleritis, a form of tuberculosis, is characterized by a slightly raised area of violaceous tint, showing no ulceration, no chemosis or adenopathy, but is tender on pressure, with considerable pain and photophobia. The condition is chronic and a local and general positive reaction to tuberculin would be obtained.

Herpes.—This is characterized by blister-like areas of the eye and forehead with severe pain.

Phlyctenule.—This is generally multiple, and is most common in children. Pain, adenopathy and chemosis are absent. Under treatment it rarely lasts over two weeks.

Gumma.—This appears as a round mass. The history of a previous chancre, or a positive Wassermann and the absence of adenopathy and chemosis, with the rapid growth, would characterize this condition. Ulceration, when present, is deep, circular and gray.

Secondary Syphilitic Papules.—I have recently had a case that showed three syphilitic papules of the bulbar conjunctiva. These resembled, in a way, the appearance of a chancre in the papule stage. The presence of other papules or evidence of such on the skin or on some other part of the body, the history of chancre, and the absence of ulceration and induration suffice to make the diagnosis.

Tumor.—Epithelioma would be most likely to be found in an old person. The slow growth of a neoplasm and the absence of chemosis and inflammation would distinguish it from a chancre.

Diphtheria.—The membrane would be extensive, the disease of from only six to ten days' duration, and the secretion abundant and sanguineous. Cultures and animal inoculation would settle the diagnosis.

Parinaud's Conjunctivitis.—This is characterized by numerous pedunculated masses of conjunctiva which later form superficial ulcers. Numerous small yellow nodules are present in the subconjunctival tissue. There is no induration of the conjunctiva. The chemosis is marked, however, and the preauricular lymph-node is swollen. This often suppurates.

PROGNOSIS AND TREATMENT

A chancre of the conjunctiva heals without permanent injury to the eye, the only evidence being a small white scar. When the lesion is located at the limbus a slight nebula of the cornea may remain; as this is not central, vision is not diminished.

The eye should be kept clean by some mild antiseptic solution. The use of calomel as a dusting-powder is recommended. Salvarsan or neosalvarsan should be given as soon as the diagnosis is made.

SYNOPSIS OF REPORTED CASES

CASE 1.—Fano,[13] 1874. Man, aged 27, cooper. Mode of infection not known. Patient gave history of having slept with a companion (syphilitic?) for seven months. Right eye was red one week. September 28 the palpebral conjunctiva was edematous. On the inferior nasal portion of the limbus was a plaque of a dirty white color, quadrangular, and the size of a lentil. This is described as being an edematous pad, of a wine-red color with a hole in the center. The cornea was slightly cloudy. The patient complained of severe and constant pain in the eye and lid, requiring the use of opium. November 7, papular eruption appeared on the side of the nose, and a discrete maculopapular eruption on the body. November 13, ulceration was reduced in size, chemosis was disappearing, and November 20 there was only slight induration and edema.

CASE 2.—Horand,[10] 1886. Man, aged 26. Bulbar conjunctiva of left eye showed marked chemosis. On the nasal side was a large red papule with an indurated base. The preauricular

lymph-node was large. Cornea free. Source of infection was probably from saliva of brother who had lues. Patient later developed papular eruption and mucous patches.

CASE 3.—Baudon,[1] 1885. Woman, married, aged 24. Disease of one month's duration. August 18, examination showed swelling of the bulbar conjunctiva below the equator of the eye, but extended all around the cornea as far as the upper culdesac. The cornea was healthy, but was surrounded on all sides by a hard resistant ring, the base of which attained the size of a cherry. Tumor was hard and covered by a white exudate, but did not have the resistance of an epithelioma. The preauricular and submaxillary glands reached the size of cherries. October 5, tumor had disappeared and patient showed mucous patches in her throat. Later her baby showed ulcerated plaques.

CASE 4.—Williams.[8] Girl, aged 18, nurse to a child with hereditary syphilis, developed chancre of bulbar conjunctiva between cornea and inner commissure. Preauricular lymph-nodes enlarged. Yellowish slough ¼ inch long, hard to the touch and adherent to the tissue beneath.

CASE 5.—Vignes,[21] 1893. Male, age not given. Patient developed two ulcers of the bulbar conjunctiva with enormous chemosis and slight secretion. Preauricular and submaxillary lymph-nodes were enlarged.

CASE 6.—Dagneloiski,[22] 1895. Female, nurse to a syphilitic infant, developed two lardaceous ulcerations in the inferior fornix and third papule on the bulbar conjunctiva.

CASE 7.—Delow,[23] 1895. Male. An elevated ulceration was present on the internal part of the bulbar conjunctiva. It had a lardaceous aspect, with cartilaginous base. Chemosis was marked. Preauricular and submaxillary lymph-nodes were enlarged. Two months later the patient developed mucous patches in the mouth.

CASE 8.—Valude and Laur,[2] 1897. J. X., aged 25, gave history of onset three weeks previous. Examination showed redness of bulbar conjunctiva of right eye on level of the nasal angle. Two weeks later a small button-like area appeared. This rapidly increased in size. Examination showed within the palpebral fissure, nasal side, about 2 mm. from limbus, a small hard tumor the size of a lentil. The form was oval, long axis vertical, base dark red, surface flat and covered by a diphtheritic exudate. Removal of this showed erosions beneath. Conjunctiva was injected, there was no pain, and only slight secretion. Preauricular lymph-node was hard and swollen, but not tender.

CASE 9.—DeSchweinitz,[25] 1898. Physician, aged 35, infected by some uterine fluid during the delivery of a child. Face wiped at the time by an attendant. Conjunctival irritation appeared six weeks later. This developed into a typical chancre of the bulbar conjunctiva. Cornea and iris were not involved. Preauricular lymph-node enlarged. "With the disappearance of the lesion on the bulbar conjunctiva and during the process of its subsidence, there developed in the upper half of the cornea a form of vascular keratitis, not unlike that which is seen in the pannus of trachoma, the blood-vessels being superficially placed beneath the epithelium, and the gray infiltrates occupying partly the epithelial layers and partly the superficial layers of the true corneal tissue. Gradually, assisted by the internal administration of potassium iodid and mercuric chlorid, the corneal lesions disappeared, and at the last examination, nine years after this condition, there was no trace of the lesion on the bulbar conjunctiva and only faint haze of the upper part of the cornea. Vision of the right eye after the correction of a mixed astigmatism was + 1.00,—2.75 c. axis 5 = 6/12."

CASE 10.—Morax,[12] 1900. Woman, aged 27. Slight ocular irritation, March 18. Examination, March 23, showed lacrimation, and enlargement of preauricular lymph-node; later a swelling appeared behind the angle of the jaw. March 29 there was acute chemosis of the bulbar conjunctiva. An area corresponding to plica semilunaris was raised, of a violaceous tint and covered by a diphtheritic exudate. This was not detached by light friction. It was in the form of an oval 6 by 4 mm. with the long axis vertical; the cornea was clear. Infection was derived from syphilitic child.

CASE 11.—Sourdille,[26] 1901. Woman, married, aged 35. Right eye. Onset ten days previous. Examination, O. D., October 18: On the bulbar conjunctiva between cornea and semilunar fold and below a horizontal line passing through the pupil were two superficial ulcerations, opal in color, each 4 by 5 mm. in diameter, and separated from each other by a bridge of conjunctiva from 1 to 5 mm. wide. The conjunctiva was wine-red. About this was an area, 12 mm. in diameter, of the consistency of cartilage. Conjunctiva was chemotic. There was no pain, photophobia or secretion. No visual disturbance. Preauricular lymph-node was enlarged. Patient treated for episcleritis. October 24, induration larger and thicker; 15 mm. in diameter; the two ulcerations had united. Conjunctiva was of a yellowish-rose color, and infiltrated so as to form a hard flesh-like mass with the cornea in the center. November 20, chemosis began to diminish. December 4, specific eruption appeared on body. Infection probably due to being kissed on the eye.

CASE 12.—Maslenikow,[14] 1901. Male, aged 27. Right eye became red one month previous. Fifteen days later swelling appeared. Examination showed at the limbus a small mass having the appearance of a phlyctenule. Eight days later this formed an ulcer surrounded by a ring of cartilaginous consistency. Preauricular lymph-node was enlarged. Later mucous patches and skin eruption appeared. Mode of infection not known.

CASE 13.—DeWecker,[3] 1865. Woman, aged 30. Ulcer on the bulbar conjunctiva, left eye, situated 0.5 cm. below and on temporal side of the cornea. This resembled a large ulcerating pustule, with a pultaceous floor; the surrounding tissues were livid; ulcer of long duration. Preauricular lymph-node swollen. Infection due to kissing on the lids.

CASE 14.—DeWecker,[3] 1873. Woman, aged 23. Indurated ulcer on the conjunctiva of the left eye, situated on the temporal side of the cornea. Preauricular and occipital lymph-nodes swollen. Infection due to kissing on the lids.

CASE 15.—DeWecker,[3] 1878. Girl, aged 6. Ulcer on the inferior and external part of the bulbar conjunctiva, right eye. Preauricular lymph-nodes were swollen. Infection due to mother using her saliva to clean child's eye.

CASE 16.—Pelissier,[15] 1904. Woman, married, aged 30. Ten days previous to examination the left eye became red. Examination, October 21, showed chemosis, with red brawny cartilaginous area, in the inferior nasal quadrant. Chemotic conjunctiva surrounded the cornea. Lacrimation present, but no secretion. Lids slightly adherent in the morning. Preauricular lymph-node was the size of a hazelnut. Husband admitted chancre (genital) two years previous. He then had mucous patches in the mouth. October 26, chemosis greater, so as to cover edge of cornea, chancre larger, size of 20-centime piece. No pain. Submaxillary and parotid glands enlarged. December 19, secondary roseola appeared on the body with redness of palate and tonsils. January 19, patient showed nasal syphilis. February, mucous patches appeared on the tonsils.

CASE 17.—Ronvillois,[5] 1909. Man, aged 24, scratched right eye while playing football. Following day eye was red and watery. No pain. Examination one week later showed marked chemosis with a red crescentic fold between limbus and caruncle. An ulcer 4 by 2 mm. with two smaller areas were present on the upper part of this fold, and a similar one on the lower part. These ulcers were yellowish and covered by a diphtheritic exudate. No secretion. V. O. D. = 2/3. No pain. Cornea clear. Preauricular and submaxillary lymph-nodes on both sides were enlarged and hard but painless. Preauricular lymph-nodes on opposite side larger than that on the side of the lesion. A roseolar rash later developed, and patient was put on specific treatment.

CASE 18.—Sbordone,[24] 1882. A physician, aged 35, one month previously developed a catarrhal conjunctivitis in the left eye. Examination showed edema of lids; eye was closed. There was abundant lacrimation, with some pain, and considerable chemosis. On the nasal side of the cornea was a cherry-like mass,

22. Delow: Cited by Pelissier (Footnote 15).
23. De Schweinitz: Personal communication.

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on which were two areas of ulceration. The margin of these were eroded and ulcerated, being made up of several pits, with the base gelatinous. Near corneal margin was a second ulcer made up from three to eight pits. Preauricular and submaxillary lymph-nodes were enlarged.

CASE 19.—Hogge,[9] 1894. A woman gave a history of having been kissed on the right eye by a luetic. Examination, O. D.: Inner half of bulbar conjunctiva was red and swollen. There was no secretion; conjunctiva chemotic. Between the cornea and caruncle, the conjunction presented a round hard nodule about 1 mm. in diameter, elevated about 2 mm. above conjunctiva. The border was badly defined, sloping to the level of the normal tissue. Top of nodule was flat and carried a slight oral depression of a paler tint (excoriation). Vision normal. Preauricular, submaxillary and cervical lymph-nodes enlarged. Fifteen days later roseola and headaches appeared.

CASE 20.—Sans Blanco,[7] 1901. Woman, aged 22. Right eye showed infiltration and chemosis of bulbar conjunctiva. On the temporal side was an area 1 mm. in extent raised from 2 to 3 mm. This was hard on palpation. On the center of this was an oval ulcer of a leaden-gray color—edges were clean to base, which was flat. No suppuration present. There was no pain on pressure. Cornea was clear, vision normal. Pre-auricular and submaxillary glands were enlarged. Two months later patient had mucous patches and was given antisyphilitic treatment. A yellowish-gray scar remained. Etiology: Husband had ulcerations of mouth and lips.

CASE 21.—Clergerie,[11] 1910. Woman, aged 26. Examination one month after onset of conjunctiva irritation. Right pre-auricular and submaxillary lymph-nodes swollen. Lids of right eye were edematous, and there was tumefaction of bulbar conjunctiva. Four days later ulceration appeared in inferior temporal portion where chemosis was most prominent. Base of ulcer was covered by granulations. Palpation gave a feeling of induration. There was no pain. Cultures showed presence of yeast. The *Spirochaeta pallida* was found in the secretion. Later a roseola appeared. Etiology: Infection evidently derived from luetic infant. About one month after assuming charge of the child the patient first noted the eye symptoms.

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ABSTRACT OF DISCUSSION

DR. ARTHUR J. BEDELL, Albany, N. Y.: In connection with Dr. Spratt's paper I should like to speak of an unreported case of initial lesion of the lid and bulbar conjunctiva. E. F. R., aged 25, a coachman, came to my office Nov. 18, 1910, giving a history of first noticing a small red pimple on his left lower eyelid about two weeks before. This was scratched with his finger-nail until it bled. Two days later the sore became more painful and, after the usual home remedies had failed, he applied for help. Ten days previously the left preauricular lymph-node became enlarged and seems to have remained the same size since. The patient had felt no pain or ache except left-sided frontal discomfort for a short time after rising. He denied lues; had had Neisser infection but no other serious illness. He had used tobacco and alcohol excessively. The right eye was negative except for a refractive error. Vision 20/30. The left eye, vision 20/50, showed congestion and swelling of both eyelids, the lower lid was indurated, especially the outer third which surrounded an irregular area of necrosis, 12 by 6 mm., with erosion through skin and conjunctiva destroying the hair follicles. The ulcer had a punched-out appearance, with irregular margin, the base was covered with a grayish-green false membrane, which was easily removed showing a grayish-blue surface which did not bleed freely. There was induration of about 2 cm. Although considerable serum oozed on scraping, the structure remained unchanged. The cornea was clear, the palpebral conjunctiva slightly injected, and the bulbar edematous with full vessels. To the temporal side of the limbus was an irregularly round area 4 by 5 mm., in opposition with the ulcer on the eyelids. The iris was not congested and the inside of the eye presented no abnormality except an oval disk. The left pre-auricular lymph-node was swollen (about 15 mm. in size) and the overlying skin was red and painful. There was no sign of skin lesion. Dr. James F. Rooney made smears of the serum and found *Spirochaeta pallida*. There was steady extension of ulceration, greater swelling of the lid and enlargement of the lymph-node even under mercury ointment. Dec. 11, 1910, five weeks after the first symptom, the patient developed a dark red maculopapular eruption on the back of the hands, a few hours later it appeared on the abdomen, then on the legs, and gradually on the face and body. December 21, he had a definite secondary fever with malaise and depression. The left lower lid was immensely swollen, although the ulcer was cicatrized except at the borders, where bleeding was easily induced. The mass had a more characteristic cartilaginous feel, the ulcer on the bulbar conjunctiva becoming smaller with much less surrounding congestion. The patient had two injections of salvarsan and made a slow but good recovery. It is interesting to give the theory as to his infection. In a stable in which he worked a fellow employee had a secondary palmar syphilid so that it seems possible that the infection could have been transferred to the reins, and later the patient using the same harness could have conveyed the active virus on his fingers to his eyelid and so infected himself.

DR. CHARLES N. SPRATT, Minneapolis: I have indirectly heard of another case that occurred in San Francisco. That makes only four cases so far reported in the United States. It seems singular that it should be so rare when lues is quite prevalent and people do not always wash their hands.

ARTIFICIAL ILLUMINATION, A FACTOR IN OCULAR DISCOMFORT *

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INTERPRETATION OF OCULAR DISCOMFORT

From the evidence at hand it seems quite certain that ocular discomfort, the result of exposure of the eyes to light, and especially that observed during close work under improper artificial illumination, must be interpreted largely as muscular fatigue of, and pain in, the extra-ocular and intra-ocular muscles, the result of their constant contraction or spasm in attempting to protect the retina from too much light. The question immediately arises: Why should this be brought about by artificial light of a certain intensity when there is no complaint made by possessors of healthy eyes from daylight illumination of several times this intensity? It seems to us to be based to a large extent on the absence of the adaptation of the eyes to a comparatively new condition. By adaptation, in this case, is not meant light and dark adaptation of the retina, but adaptation of the eyes by process of evolution to a new environment.

EVOLUTION OF THE EYE

Evolutionary changes in any organ of the body are brought about by a change in the actual requirements of that organ, the result of different conditions and environment extending over a very long period of time. There was practically no long-continued use of the eyes under artificial illumination before the discovery of the refining process, which produced an efficient artificial illuminant from petroleum about the year 1854.

* Because of lack of space this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints. A copy of the latter will be sent by the author on receipt of a stamped addressed envelope.

Since Edison's production of the carbon incandescent lamp in 1878, the number of hours the eyes are used for close work under artificial illumination has been enormously increased. This increased use of the eyes has been made possible by the development of artificial illumination and the improvement made in it. Thus it must be apparent that far from sufficient time has elapsed for evolutionary changes even to begin to adapt the eyes to the tremendously increased use and comparatively abrupt change in environment. The eye, therefore, by process of evolution should still be best adapted to the energy values of the wave-lengths of solar light.

EDRIDGE-GREEN'S THEORY OF VISION

How the visual act is accomplished is not absolutely proved, but the theory advanced by Edridge-Green[1] appeals to us as being the most rational. The hypothesis advanced to account for ocular discomfort from artificial light in this paper will be based on Edridge-Green's theory.

EFFECT OF LIGHT ON VISUAL PURPLE

The result of investigations of the photochemistry of the visual act points to the existence within the retina of an unstable, decomposable photochemical substance, the visual purple, which does not cause nerve excitation as long as it remains unchanged. It has been proved beyond all reasonable doubt that this material is broken up or decomposed by the action of light. Its distribution in the ocular apparatus is in that portion where there is every reason to locate the effects of light excitation, that is, the visual cells. The moment this substance is decomposed by the action of light it gives off products which must be regarded as exciters of vision. The action of light is direct. Protected areas on the retina remain unaffected. The rapidity of bleaching or decomposition of the visual purple depends on the intensity and color of the light. The sensitivity curve of the eye for luminosity at ordinary intensities shows a maximum effect in the yellow and green portion of the spectrum. (See curves a, b and c, Fig. 1). It is a well-known fact that the luminosity curve of the eye varies with the intensity of the light, shifting toward the green with illumination of low intensity and toward the orange and red with illumination of high intensity. (Fig. 1. Curves a, b and c).

Luckiesh[2] has determined that visual acuity, for a reading distance of 33 cm. with monochromatic light, is greatest in the yellow green of the spectrum. Thus, it would seem that the radiant energy which has the greatest decomposing action on the visual substance is productive of the greatest visual efficiency, that is, the yellow green waves.

PROTECTIVE MECHANISM OF THE EYE

Inasmuch as the action of light is direct and the decomposition of the visual purple depends on the intensity of the physical characteristics of the light, gaining entrance into the eye, the end elements of the optic nerve must have some means of protection from the varying degrees of intensity of illumination to which they are constantly subjected.

The protective mechanism of the eye consists of the brow, the lids, and iris. There are also two protective agents within the retina itself: the pigment epithelial layer of the retina and the visual purple.

1. Edridge-Green, F. W.: Color-Vision and Color-Blindness, Hunterian Lectures, 1911, Lancet, Feb. 4, 1911.
2. Luckiesh, M.: The Dependence of Visual Acuity on the Wave-Length of Light, Electrical World, 1911, No. 58.

impulse is set up which is conveyed to the visual center.

When a ray of light enters the eye a centripetal and the motor center of the eye from which centrifugal impulses are sent to the various points of distribution of the motor oculi nerve. Naturally the greater the intensity of the retinal stimulation the greater the intensity of the centripetal and centrifugal impulses.

SPECTRA OF DAYLIGHT AND COMMON ILLUMINANTS

Ives[9], on striking an average of a large number of spectrophotometric measurements on various kinds of daylight, concluded that the color of average daylight "may be accepted as having very nearly the energy distribution of a black body at 5,000° absolute," while the ordinary illuminants, such as the ordinary 16 candle-power carbon incandescent lamp, the tungsten incandescent lamp and the Welsbach gas burner, have the

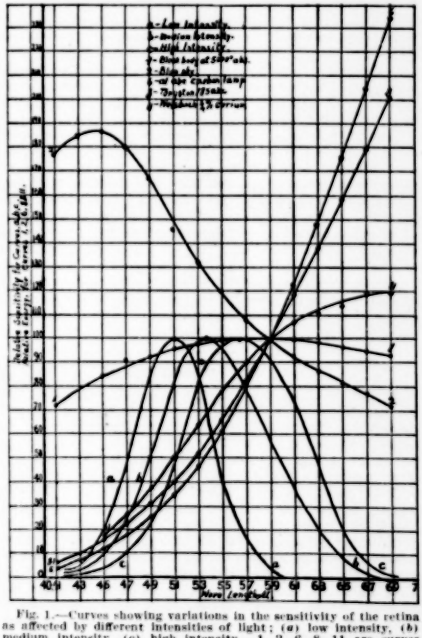

Fig. 1.—Curves showing variations in the sensitivity of the retina as affected by different intensities of light; (a) low intensity, (b) medium intensity, (c) high intensity. 1, 2, 6, 8, 11 are curves showing different energy values by wave lengths from different commercial lighting sources and daylight; (1) black body at 5000° abs.; (2) blue sky; (6) 3.1 wpc. carbon lamp; (8) 1.25 wpc. Tungsten lamp; (11) 0.75 per cent. cerium Welsbach.

color of an incandescent black body heated to 2,000° C. This means that when giving the same measured illumination, our common illuminants, as compared with daylight, are very deficient in blue light and have an excess of yellow and red light. (Fig. 1, curves 1, 2, 6, 8, and Table 1).

In comparing these it will be seen at a glance that the wave-lengths from that portion of the spectrum which cause the greatest decomposition of the visual purple

9. Ives, H. E.: Color Measurements of Illuminants; A Résumé, Tr. Illum. Engin. Soc., 1910, No. 5; Subtractive Productions of Artificial Daylight, Electrical World, 1911, No. 57.

V. 6
1913

(the greenish yellow waves) are more nearly equal in energy than in any other portion of the spectra. There is in the spectra of artificial illuminants a marked increase in the energy toward the red end and a marked decrease toward the blue end compared with the spectrum of solar light.

HYPOTHESIS AS TO CAUSE OF OCULAR DISCOMFORT

The result is a very marked difference in the quality of the retinal stimulation from artificial illumination compared with that of solar light. *Because sufficient time has not elapsed for evolutionary changes to take place and adapt the eye, the visual apparatus cannot be subjected to this difference in quality of radiant energy for any length of time at proportionately as high intensities with artificial light as with daylight without manifesting ocular discomfort.*

The hypothesis advanced is that with light waves from artificial sources after reaching a certain intensity, which is probably slightly above that which is ordinarily required for general use of the eye, the difference in the spectral quality of artificial light, compared with that of daylight, causes a more rapid decomposing of the visual purple, with consequent increased rapidity in its exhaustion and increased stimulation of the nerve centers controlling the protective mechanism of the eye; there is also a lessened opportunity for reproduction of the visual purple as the intensity of the light source is practically constant. We must not forget also that the eyes are used, especially for close work, a very great many more hours by this generation than the previous one, and the question of ocular fatigue, pure and simple, must be taken into consideration.

Fig. 2. Fig. 3. Fig. 4.

Fig. 2.—Graphic expression of the law of specular reflection.
Fig. 3.—Graphic expression of the law of diffused reflection.
Fig. 4.—Graphic expression of the law of spread reflection.

SYMPTOMS OF OCULAR DISCOMFORT

One individual may be affected differently from another. The most common complaint, after a longer or shorter period of exposure, is that the eyes feel hot and dry with a sense of fulness about them. The details of objects looked at become a little blurred, as if the illumination were being diminished. At this stage winking is noticeably increased, as is also the hot, dry feeling and sense of fulness about the eyes. There is an occasional stinging pain causing the lids to be tightly closed for a few seconds. Later, if the effort to use the eyes is persisted in, the finer details of objects are practically lost for an instant and can be brought out only by closing the eyes for a few seconds. There is frequently an actual ache in the eyes and head, mental activity is dulled, and there is an almost complete inability to keep the eyes open, together with marked drowsiness. The eyeballs and lids usually show more or less congestion. A short rest with the eyes closed or shaded will bring about a marked sense of relief, which on resuming the occupation gives place to the same train of symptoms that now make themselves manifest much more quickly. Increasing the intensity of the illumination for a short time will seem to give relief, but it is very quickly followed by greater discomfort.

EFFECT OF INVISIBLE RADIATION

That this effect is not due to the ultraviolet rays is practically proved by the experiments of Parsons,[11] Birch-Hirschfeld, Behr,[12] Fuchs,[5] Schanz and Stockhausen. The cornea and lens absorb practically all the rays of less than 400 microns wave length.

It would seem from the result of recent experiments reported by Vogt[14] that the effect of the ultrared rays must be taken into consideration. The consensus of opinion however is that the harmful effects come from luminous radiations of greater than 400 microns wave length and less than 700 microns. Fuchs says:

> The disagreeable sensation of dazzling by bright light is produced solely by the visible rays of the spectrum and it is these rays that are damaging to the eye in case of affections of the retina and chorioid.

SUPPLEMENTARY FACTORS CAUSING OCULAR DISCOMFORT

Muscular contraction demands an increased blood-supply to the parts and we have a passive hyperemia which in a short time becomes active, the result of muscular spasm. This is manifested as a hot, dry feeling and a sense of fulness or sensation of "sandiness," which is accentuated by the lid rubbing on the eyeball, the result of increased winking. The intensity of the artificial illumination is practically constant and does not vary between such wide limits as does the intensity of daylight, so there is less opportunity for regeneration of the visual purple. After a time the supply of this photochemical substance becomes materially decreased; the decomposing activity of the light remaining the same, the resultant energy of the cone stimulation is diminished because of the exhaustion of the visual purple. The result is a lessened centripetal impulse with consequent lessened centrifugal impulse, a relaxation of the muscular action of the protective mechanism and the already overtired eye is flooded with light. This causes a spasmodic, very active efferent and afferent impulse, which is manifested by a stinging pain, causing the eyes to be tightly closed. After there has been sufficient restoration of the visual purple the eye can again continue its work. With daylight there are continual changes in intensity which tend to produce more restful ocular conditions.

SEVERE AND LASTING INJURIES

There is no doubt that the severe and occasionally lasting injury to eyes exposed to the arcing of high intensity electric currents, and from viewing a solar eclipse, is the result of the action of the intense light on the naked retinal elements. The pupil is caught unawares and neither the lids nor the iris have time to act in protecting the eye. The intensity of the light decomposes the visual purple so rapidly that it leaves the rods and cones unprotected, and, before fresh supply can be rushed to their protection or the pigment epithelium can throw its protective extensions forward between the rods, the damage is done.

11. Parsons, J. H.: Some Effects of Bright Light on the Eyes, THE JOURNAL A. M. A., Dec. 10, 1910, p. 2027.
12. Behr: Alterations and Injuries of Eyes Through Light, Arch. f. Ophth. (Graefe's), 1912, lxxxii, 509.
14. Vogt, A.: Experimental Researches on the Penetrability of the Transparent Media of the Eyes for Ultrared Rays from Artificial Light, Arch. f. Ophth. (Graefe's), 1912, lxxxi, Part 1; Some Measurements on Penetration of Heat Rays in the Human Eye as Well as the Upper Lid, Together with Some Observations on the Biologic Working of Ultrared Light, *ibid.*, 1912, lxxxiii, Part 1.

INSUFFICIENT ILLUMINATION

Insufficient illumination is a factor producing ocular discomfort, especially in work requiring the observation of fine detail. The intensity of the light rays is not great enough to cause a rapid breaking up of the visual purple and consequent cone stimulation. The eyes are brought closer to the object in the effort to see more clearly, which results in an increased effort of the muscles of accommodation and convergence with consequent ocular fatigue. The pupil in weak light dilates to admit a greater amount of light. The accommodative act is accompanied by a contraction of the pupil, and its attempts to dilate under the conditions of illumination and contract under the accommodative effort add to the ocular discomfort.

GLARE AND ITS CAUSES

There is, too, another grave cause of ocular discomfort under illumination by either natural or artificial light, but especially under the latter, on account of its misuse through sources of too high intrinsic brilliancy, too high intensity, or with too little diffusion. This cause is commonly known as "glare" and appears to us in many forms and through many causes.

1. We may, for instance, have glare due to the intrinsic brilliancy of the sun, the Welsbach mantle, of the incandescent lamp filament, or any other form of illuminating unit when the source appears approximately within the line of vision.

2. We may also have glare in a very similar way due to the total light entering the eye from a source whose intrinsic brilliancy is, relative to the source cited above, comparatively low, as for instance, any one of the units mentioned when enclosed in a diffusing ball or situated behind diffusing glass, or from the sky area—blue or overcast—directly in the line of vision, but remote from the sun.

3. We may also have a very objectionable form of glare by reflection from surfaces such as paper from which we read or on which we write, varnished desk tops, lightly tinted walls, glass in picture frames and other similar conditions of every-day occurrence.

REMEDIES FOR OVERCOMING GLARE

The remedies for overcoming the glare due to the first two causes are practically effective and easily available. The ill effects can be minimized, for instance, by enclosing the sources in diffusing mediums which will cut down the intrinsic brilliancy of the unit; by subdividing units for any given intensity of illumination in an installation into small units, enclosed as above; by distributing units more or less uniformly over the area to be illuminated, instead of concentrating them at one point, thus avoiding the possibility of any great number occupying positions within the line of vision at any one time; by placing the units high above the natural line of vision, or concealing the source entirely from view by means of opaque reflecting mediums, as in the indirect system, thereby transferring the phenomenon from the class under discussion to the third class.

GLARE BY REFLECTION

It is desired at this time to call particular attention to glare by reflection. As by far the major portion of our visual functions are performed through the medium of reflected light, this form of light is, from a practical point of view, by far the most important. It is believed that until recently there has been no real appreciation of the magnitude of damage done by reading from pages with highly calendered and glazed surfaces on which printing is done by means of equally brilliant ink. We see the printed page by virtue of reflected light only and this is dependent on the illumination superimposed on its surface and on its reflection characteristics. This will be more apparent on consideration of the principles of reflection.

LAWS OF REFLECTION

Probably the only conception which most individuals have of the phenomenon of reflection is based on the extremely simple and therefore easily remembered law taught for years: "The angle of reflection is equal to the angle of incidence." This law is as true to-day as in years gone by if applied to the surfaces to which we were taught to apply it. Like many other physical laws, it is correct within its limitations, but must be expanded to-day to meet the newly appreciated conditions. The law, as stated above, is perfectly true and accurate when the light impinges on surfaces such as mirrored glass, polished metal or other surfaces approaching the same characteristics—in other words, it applies accurately to *specular reflection* (Fig. 2). A beam of light, under this law, is reflected, minus loss by absorption, in a single direction, depending on the angle at which it strikes the surface.

As our reading surfaces are neither metal nor glass, and yet seeing the page depends on reflected light, it will readily be seen that other types of reflection must exist. If the surface is irregular, or mat, as in the case of blotting-paper, paper with the desirable characteristics discussed in the reference given below, and calcimined ceiling and wall surfaces, the reflected light will, more or less perfectly, obey the law of *diffuse reflection* (Fig. 3), according to which the incident beam will be broken up and diffused, or scattered, in all directions, the maximum rays of reflected light taking a direction perpendicular to the reflecting surface; thus, if the diffusion is perfect, dropping straight down from the ceiling, for instance. The intensity of the rays in any other direction is proportionate to the chords of a circle drawn tangent to the surface at the point of incidence.

Some surfaces, such as mediums which are in their usual state specular in character, but which have been depolished similarly to a ground glass surface, obey a third form of the law of reflection—sometimes called the law of *spread reflection* (Fig. 4), in which there is only a partial diffusion, or scattering, of the incident beam of light the maximum reflection taking place at an angle assigned by the law of specular reflection. The intensity of the reflected rays in any direction may thus be represented approximately by an ellipse, whose axis is in the direction of the angle of specular reflection and which is drawn tangent to the surface at the point of incidence.

The preceding argument is extracted from a more or less complete discussion of this form of glare contained in a paper[15] which was read before the Chicago section of the Illuminating Engineering Society February 22, which, in turn, was prompted by a most thorough and complete development of this subject in a paper by Mr. Arthur J. Sweet.[16]

DAYLIGHT ILLUMINATION

In the discussion up to this point particular stress has been laid on the disadvantageous qualities of arti-

15. Vaughn, F. A., and Black, Nelson M.: Discussion of Ocular Comfort and Its Relation to Glare from Reflecting Surfaces. Electroforce, April, 1913.

16. Sweet, Arthur J.: Reflecting Mediums, Railway Electric Engineer, January, 1913.

V. 6
1913

ficial illumination and their effects on ocular comfort and fatigue. In our enthusiasm over the pursuit of this phase of the subject, however, we must not lose sight of the fact that daylight also has deleterious characteristics which often produce very uncomfortable and harmful results on the ocular organism.

NATURAL ILLUMINATION FAR FROM IDEAL

Daylight has been assumed by many in the past, and by the laity especially, to be the perfect illumination. This idea, it is believed, an unbiased consideration of the subject will dispel. Its principal advantages are its abundance and its cheapness; its color may or may not be an advantage, according to results of future investigations. Its disadvantages are certainly very apparent in its wide variations in intensity and diffusion—far exceeding the limits placed by public service commissions on electric lighting and gas service; its excessive quantity, or overabundance, based on what has been demonstrated to be sufficient artificial illumination; its excessively glaring effects when an attempt is made to utilize it unscreened or undiffused; its undependability on account of weather conditions; its inability to supply us with twenty-four-hour service, which service is demanded of almost every public utility company in this country; and also—for which man alone is to blame—its incorrect application to and utilization in our interiors.

It is, perhaps, rather an egotistical statement, but illuminating engineers to-day believe that they can produce far more perfect illumination than daylight as far as regulation, control of intensity and color, continuity of service and some other features are concerned, and it is believed that they will be able to demonstrate it as time goes on. Therefore it is desired to call attention to the imperfections of daylight illumination and attempt to eradicate, or at least adjust, the idea that daylight is perfect illumination.

It is not deemed necessary to enter into the analysis of the composition of daylight at this time, or to more than call attention to the fact that in "daylight," besides the visible rays, there are rays in the infrared region which present themselves in the form of heat, and in the ultraviolet region, those which have well-recognized chemical powers. These extreme rays are probably not useful in producing vision and therefore, if allowed to enter the eye, may be deleterious in action. It is well known, in fact, that the eye is so constructed as to protect itself, to a marked degree, against their action, and some of the disorders of the ocular mechanism which are the results of exposure to solar light are undoubtedly due to their presence. As Alger[17] says, for instance:

> In snow and desert blindness the light is broken up by reflection from the crystalline snow or sand and the actinic waves produce intense inflammation of the conjunctiva which, if long continued, results in total disability. Even in temperate climes one suffers more or less from glare and burn from direct or reflected sunlight.

It will be seen from the preceding discussion, we believe, that it is not by any means an established fact that daylight illumination is ideal and these comments are made for the purpose of calling attention to its imperfections, and to the necessity for scientists and engineers to adjust the conditions surrounding our every-day use of natural light so as to protect the eye, as completely as possible, from its bad effects.

17. Alger, Ellice M.: Illumination and Eye-Strain. Tr. Illum. Engin. Soc., 1913.

Of course, notwithstanding the above criticism, we must all be very thankful for the abundance and cheapness of the illuminant supplied us by Nature, but should show our appreciation by learning how to utilize it.

The task of illuminating engineers in controverting the idea which is so prevalent, that sunlight is the ideal light, is fully appreciated. It is true also that, as a general proposition, it is our utilization of the daylight in our rooms which is most harmful. It usually comes in from a horizontal direction and therefore, in natural illumination as we generally utilize it, we have decided non-uniformity, insufficient minima and excessive maxima, as well as the undesirable direction of the light, and deep contrasts between the window spaces and dark areas.

INFLUENCE OF DIRECTION OF LIGHT ON OCULAR COMFORT

While speaking of the direction of the light it might be mentioned that the snow and desert blindness are greatly aggravated by the unnatural and upward direction of the light from the ground. For this reason protection is realized by spectacles made of disks of leather with holes partially cut out and bent down so as to intercept the light from below, and by blackening the skin below the eyes to obviate upward reflection of light coming from above. It may therefore be that the bad effects of excessive light, reflected from reading surfaces and desk tops, may be considerably accentuated by its upward direction under certain conditions — the light striking portions of the retina not naturally equipped to receive it harmlessly.

CONCLUSION

In conclusion it might be said that the eye at present, through the process of evolution, is adapted to solar light and can accommodate itself to marked variations in intensity of light of this character with less discomfort than with artificial illumination, because of the quality of the retinal stimulation. There is, however, no reason why ocular discomfort should be any more in evidence with the use of artificial illumination than with solar light, when due consideration is given to the various factors and important conditions bearing on its proper and intelligent application; for, as has also been pointed out, unintelligent and unscientific application of solar light to our every-day needs may and does produce even more harmful results than those often produced by artificial illumination. Beneficial results can certainly be obtained through thorough investigations of the effect of different wave lengths, and amounts of energy on the eye; the adjustment of the color of artificial illumination by selection and design of source; the modification of the effects by selection of color and characteristics of the surroundings; the change in color of reflected light by the color absorption characteristics of paper, ceiling and other surfaces and the practical application of the ultrared and ultraviolet absorption characteristics of glass and other media. The ocular comfort of this and future generations depends on the solution by illuminating engineers of the problems of the proper application of artificial and natural illumination and it is gratifying to note the concordance of ideas and methods of application, and the unanimity of purpose, exemplified by the work of the active members of this profession, in cooperation with physiologists, psychologists and ophthalmologists who are interesting themselves in this subject.

ABSTRACT OF DISCUSSION

MR. HERBERT E. IVES, Philadelphia: For progress in this line it is essential to have the cooperation of the ophthalmologist and the engineer. Whether this cooperation takes the form of joint action with the Illuminating Engineering Society through joint committees or through the cooperation of individuals, as in the present case, matters little, but the two points of view must both be considered if artificial light is to be rendered free from danger to the public. The Illuminating Engineering Society was founded for the study of the production, measurement and utilization of light. It started out as a body of electrical and gas engineers. Later we have added physicists, architects, ophthalmologists, physiologists and psychologists. What we have accomplished may be described as objective, what we have not, as subjective. We have analyzed light into what we consider its important elements—color, intensity, direction and diffusion. I feel safe in saying that, given the money and a fire-proof laboratory, we can produce any conceivable sort of illumination. We can copy daylight, we can produce light containing no heat-rays, light containing no ultraviolet, light coming from above, from the side and from visible or concealed sources. We are well equipped so far as the physical analysis and synthesis is concerned. When we turn to the subjective side, however, we meet limitations. The eye is the ultimate consumer and we have learned by sheer experience that the greatest engineering efficiency is not the greatest visual efficiency. An engineer would expect the most efficient illumination of a printed page to result from pouring all the available light on it. We find, however, that it is much better to put some of the light on the surroundings, thereby lessening contrast and preventing ocular fatigue. It is then that we need the ophthalmologist. We must know about the eye and we have no means of testing to know whether an illumination is affecting the eye beneficially or detrimentally. Specifically, we need test methods. The classic tests for ocular efficiency are woefully inadequate. Visual acuity, perception of brightness-difference and color-difference perception are altogether too crude. Their indications are not definite enough to distinguish between illumination conditions so extreme that mere inspection would condemn one as acutely uncomfortable and commend the other as admirable. We look to the psychologist and the ophthalmologist for new and scientific means of determining when the eye is abused. Reciprocally, the light specialist can be of assistance in this work to the ophthalmologist. He can insure that the various physical factors are viewed in their right proportions. He can keep the ophthalmologist from "barking up the wrong tree." For instance, he can, from his physical analysis of light, point out that there is no significance to a discussion of the relative comfort to the eyes of oil, gas, electricity or daylight. The manner of production of light is a mere accident. The real factors are intensity of illumination, color of light, the intrinsic brilliancy of the visible bright surfaces (of light source or illuminated object) and the direction and diffusion of the light. The much-lauded student-lamp can be exactly copied by gas or electricity. It is a light source of rather low intrinsic brilliancy, of yellow color, concealed from the eye and so made that it must practically be used as a side-light source. If it has great merit, it is entirely in these copiable factors, not in its fuel. The lighting expert can also inform the ophthalmologist that the ultraviolet light in ordinary light sources is far less by actual measurement than in a corresponding amount of innocuous daylight. He can point out that practically all artificial light sources have an enormous excess over daylight of infra-red, or heat-radiation and that here is the logical point of study, rather than the greatly advertised ultraviolet. Perhaps the greatest pitfall for the non-engineering student of lighting is the common confusion between illumination and intrinsic brilliancy. Do not confuse a bright light with a high illumination; they may or may not go together. A tungsten filament of 1,000 candle power per square inch gives as bright an illumination on the table if an eye shade is worn but the difference in comfort between seeing the filament and not is enormous. Perhaps I can make more clear the present state of illuminating engineering and its needs by an illustration of the only method of test we have for visual efficiency. This is what lawyers call the case system. If we find an illumination system that looks good we analyze and copy it. The danger is that we will copy a lot of non-essentials. Why, it will be asked, do we not then simply copy pleasant daylight illumination from windows, proved good by experience? When daylight from windows is analyzed it is found to be illumination chiefly from a large area low intrinsic brightness concealed light source (the sky) entering at the side and producing a range of visible brightness of about 200 to 1. By night we light the same room by a small visible, high brightness source from above, 95 per cent. of it heat-radiation, producing a range of visible brightness of 100,000,000 to 1. I have recently calculated that accurately to copy daylight in a room, thereby changing in this radical manner the method of artificial lighting, would involve spending more for the lighting of one room in a night than the rent of the whole house for a week. The illuminating problem is, therefore, one of the conservation of natural resources as well as the conservation of vision. It is necessary to know what the eye must have for health and what it can dispense with. Only by the cooperation of the ophthalmologist and the engineer can this be brought about.

SOME COMPARATIVE MEASUREMENTS OF THE SKULL AND SELLA TURCICA

WITH REPORT OF EIGHT CASES *

JOHN B. POTTS, M.D.
OMAHA

For many years ophthalmologists have observed ocular affections which were caused by disturbance of the pituitary body. These had been reported and correlated so that a set of rather definite ocular symptoms had been established, but owing to the inaccessibility of the gland little progress had been made in alleviation of the trouble. It remained for Erdheim, Oliver, Schafer, Herring, Fröhlich, Borchardt, Goetsch, Cushing, Jacobson, and others to determine the anatomy, physiology, pathology and symptoms so that a fair understanding of the function of the gland and its disorders was obtained. In view of the intimate relation between the pituitary body and the optic nerve, it seems probable that a large number of these cases will be first seen by the ophthalmologist in the future, as they have been in the past. The subject is only in its infancy, and I think that there are many important points in diagnosis which as yet we have only surmised.

SYMPTOMS

It is impossible for me, in this paper, to do more than mention some of the more prominent symptoms, and for more detailed information on the subject I would refer you to Cushing's book.[1] Three general types are recognized:

1. Hypopituitarism, of Fröhlich's type, due to a lack of development or secretion; the pituitary body either remains small or its secretion inadequate. Hence we have persistence of infantile skeletal growth, adiposity, absence of hair, sexual infantilism. This is pretty well recognized and is not of the type that causes disturbance of the optic nerve.

2. Typus Launois. Acromegalia, hyperpituitarism during adolescence, with symmetrical increase in the skeletal growth, so-called giantism.

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* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

1. Cushing, Harvey: The Pituitary Body and Its Disorders, Philadelphia, Lippincott, 1912.

3. Typus Marie. Hyperpituitarism in adult life resulting in enlargement of the bones of the head and face, and lengthening of the bones of the extremities.

It is the third class that we most frequently meet in our work, that is, cases in which there is an enlargement of the gland and the patient consults us for headache, disturbance of vision, muscular paresis of the eye, exophthalmos, etc. Subjective symptoms are headaches, lassitude, inability to fix attention, loss of memory, anosia, slimy discharge from the nasopharynx, constipation, etc.

Skeletal Symptoms.—In the early stages there may be an increased muscular strength, later this is followed by a diminution of strength, which often becomes very extreme.

Sexual Symptoms.—In the early stages there may be a libido sexualis followed by impotence in the male, and cessation of menses in the female. Other symptoms of lack of glandular activity are increased carbohydrate tolerance, adiposity, polyuria, dry skin, which may be rough or scaly, lack of perspiration, loss of hair, especially on the body.

Eye Symptoms.—Recurrent periods of temporary blurring of the vision with a gradual loss of sight. The fundus usually appears normal except for primary atrophy of the optic nerve, although in some cases there may be swelling and edema of the nerve-head and hemorrhages into the retina. The fields are often very characteristic, although there may be many variations due to the fact that the tumor does not enlarge symmetrically. The typical fields are a bitemporal hemianopsia which first appears in the upper outer quadrant. The fields for color are generally affected before those for white.

Roentgen-Ray Findings.—Normally the sella turcica is very clearly outlined; clinoid process above and anterior, sphenoid cells anterior and below, and the body and the posterior clinoid process below and posterior. If the enlargement of the gland is only moderate we may see the sella with its walls much distended, but the different anterior structures still present. If the extension is anterior, the clinoid processes are gone. If downward, the floor of the sphenoid is forced into the sphenoid cells, and if backward the posterior clinoid process is obliterated. If the growth has become sufficiently large all these conditions may be present, and it is impossible to see any of the normal outlines of the sella.

In order to have a clear understanding of the pathologic changes that have taken place in the skeletal outlines it is necessary to have a well-established idea as to the normal conditions. For the past four years we have been using the Roentgen ray to localize foreign bodies and suspected foreign bodies in the eye. These patients had no symptoms of pituitary trouble, and the age varied from 4 to 60 years; hence a comparison of these plates should give us a fairly accurate idea as to the Roentgen-ray picture presented by a normal sella turcica. In addition to this I have found twenty-nine plates in which it was possible to secure the measurements from the anterior clinoid processes anterior to the inner table of the frontal bone, also the perpendicular measurements from the anterior clinoid processes to the inner table of the dome of the skull. An analysis of these tables shows that the normal pituitary body varies from 8 mm. anterior-posterior to 13 mm., and from 6 mm. in depth to 10 mm. While a measurement greater than this may not be pathologic, it seems to me that it should be looked on with suspicision. A glance at the table of the measurements of the skull shows at once a marked uniformity, in contrast to the measurements of those of the pituitary body, and it seems to establish conclusively that there is no apparent relation between the size of the latter and the former. Another striking point is that, in contradistinction to the frontal sinuses and sphenoids, the sella turcica reaches a size nearly equal to that of an adult at an early age, and, as will be clearly shown in the illustrations to follow, there seems to be no relation as to size between the different sinuses and the sella turcica. These observations emphasize the fact that the pituitary gland is, like the brain, of vital importance to the development of the body, and the skeletal growth makes an early provision for its proper housing.

REPORTS OF CASES

I wish now to report the following cases, seen jointly by Dr. Gifford and myself:

CASE 1.—J. F. McD., aged 26, came to us Sept. 10, 1912, on account of slight failing in right eye; no headache, dizziness or nausea. He had had the ordinary diseases of childhood, but had been otherwise healthy. About one week before consulting us he noticed that he did not see well in the right eye. When questioned he admitted that he had been growing for three or four years, and his height had increased from one to two inches.

Inspection: Man of unusually robust appearance, height 6 feet 1 inch, weight 180. Well muscled and had been, up to this time, of unusual strength. Hands and feet noticeably large. Hirsuties normal; no enlargement of other ductless glands. Vision, right eye 20/200, left eye 20/20, not improved by glasses. Ophthalmoscope: Fundus normal, temporal fields somewhat constricted. Nose thin; serous discharge over posterior part of left middle turbinate and nasopharynx.

TABLE 1.—MEASUREMENTS OF THE SKULL AND PITUITARY BODY IN TWENTY-NINE CASES OF NORMAL HEADS*

	Measurements Skull		Measurements Sella turcica			Measurements Skull		Measurements Sella turcica	
	A. P.	P.	A. P.	D.		A. P.	P.	A. P.	D.
	cm.	cm.	mm.	mm.		cm.	cm.	mm.	mm.
1.	6	10	10	10	16.	6.25	10.25	11	9
2.	6	9.5	12	8	17.	5.5	9.5	10	5
3.	6	10.5	11	7	18.	6	9.25	10	9
4.	5.5	9.5	10	6	19.	6.5	9.5	10	6
5.	5.5	9.25	12	9	20.	6.5	10	11	7
6.	6.5	10.25	13	10	21.	6.5	10.5	10	8.5
7.	6.25	10.25	12	10	22.	6	10.5	12	10
8.	6	9.5	12	8	23.	6.75	10	10	8
9.	5.5	9.5	12	7	24.	8	10.5	10	8
10.	6.5	11	11	9	25.	5.75	10	12	10
11.	6.25	11	14	10	26.	5.5	9.25	8	10.5
12.	6.75	9.75	12	8	27.	6	9.5	10	8
13.	7	11	12	8	28.	6	10.5	11	6
14.	5.75	9.25	10	6.5	29.	6.5	9.5	10	8
15.	6.5	9.75	12	9					

*In this and the following tables, under "skull," "A. P." refers to the measurements from the anterior wall of the sella turcica to the internal table, at a point opposite the frontal prominence. "P." stands for a measurement from the anterior clinoid process perpendicular to the inner table of the skull. Under "sella turcica" "A. P." refers to the anterior-posterior measurements of the sella, and "D." to the depth of the sella turcica. From a careful examination of the table it will appear that there is no definite relation between the measurements of the skull and the pituitary body.

Sept. 19, 1912: Sent to hospital, under treatment of rest and salicylate. Vision of right eye returned to 20/20 minus 2. Sent home.

Oct. 22, 1912: Reported for inspection; vision, right eye 20/40, left eye 20/15. Ophthalmoscope: Right disk a little pale. Sent home with letter to doctor and instructions to take potassium iodid, 20 grains, three times a day.

Nov. 3, 1912: Returned; condition decidedly worse, vision right eye 20/30, left eye 20/20. Ophthalmoscope: Right disk slightly paler than left; fields becoming more constricted. Headache and peculiar full feeling in head accompanied by slight dizziness and some nausea. Gait becoming noticeably ataxic and patient complains of great loss of strength; knee-jerks lessened. Carbohydrate index not noticeably increased, no glycosuria or polyuria. Roentgen ray shows a large sella turcica, anterior posterior 15 mm., depth 10 mm. Skull anterior measurements 6.25 cm., perpendicular measurements 9.5 cm. Bones of forearm and leg and phalanges decidedly lengthened, and characteristic epiphyseal changes, described by Cushing and others.

Diagnosis: This I consider to be a typical case of acromegalia coming on during adult life. I was not able to secure any photographs and hence could make no comparison

as to any change in features, but the lips had not become thick and, except for a possible slight exophthalmos of the right eye, patient said that there was no noticeable change in his appearance. He refused operation and passed out of our hands; we have not seen him since.

CASE 2.—D. M., aged 40, Dec. 30, 1912, consulted us because she noticed vision failing and on account of severe temporal headaches. She had been a healthy child and had no serious illness until ten years ago she had a nervous breakdown in the fall following mother's death and financial reverses. In the following spring she says she was treated for rheumatism. About this time she began to have headaches, some dizzy spells, serious vomiting spells and terrific temporal headaches; she felt exhausted and had no ambition to do any work. She has not menstruated since trouble began ten years ago, at age of 30. About the same time she noticed that she was gaining in flesh and has kept on gaining in weight until about two years ago. Since then she has been feeling somewhat better and has not gained in weight until this spring.

Mother died ten years ago, tumor of the stomach, aged 67. Father died aged 44, cancer of the stomach; patient had two brothers, one living older than sister, normal; the other died of typhoid fever.

Inspection: Rather a comely woman, intelligent and refined face, height 5 feet 8 inches, weight 170, no enlargement of head, hands or feet, skin dry and smooth.

I was not able to obtain carbohydrate tolerance.[2]

Vision: Right eye 20/70, left eye 20/200; glasses do not help. Ophthalmoscope: Right eye nerve-head a little pale, left eye outer half pale and excavated. Fields, left eye typical temporal hemianopsia, right eye temporal fields not so much restricted as left. Nose normal. Roentgen ray: Outlines of

TABLE 2.—MEASUREMENTS OF THE SKULL AND PITUITARY BODY IN WHICH THERE WAS SOME INVOLVEMENT OF THE PITUITARY GLAND

	Measurements Skull		Measurements Sella turcica	
	A. P. cm.	P. cm.	A. P. mm.	D. mm.
1.	7	.....	18	16
2.	5.25	.....	26	16
3.	6	.....	..	9
4.	5.25	9.25	(gone)	
5.	6.5	.....	10	7
6.	6.5	10	13	10
7.	5.5	10.25	14	10
8.	6.25	9.5	15	10
9.	6	.....	15	10
10.	6	11	15	10
11.	6	9.5	9	16

sella entirely obliterated, floor occupying most of sphenoid cells, anterior-posterior diameter 26 mm., depth 16 mm.

Diagnosis.—Strumous enlargement of the pituitary gland with deficiency of secretion of posterior lobe.

Operation was advised, but patient had planned a trip to the West and insisted on making this before having anything done. I saw her only once and have no report since.

CASE 3.—E. C. O., aged 46, consulted us Feb. 21, 1912. His sight had been failing for some time past; he had had headaches in temples and dome of head for past eight or ten years. "Says he has catarrh. Chronic, persistent constipation. Has not perspired for several years. Hair (face, head and body) very scanty; formerly had heavy beard. Is very fond of sweets, and carries candy around in his pocket much of the time. Small-boned and says he was slim until about ten years ago, when he began to put on flesh. Now quite heavy with pendulous abdomen. Family history negative. Mentality, answers readily but mental faculties seem to work slowly. Says he is inclined to sleep more than formerly and often feels drowsy during the day."

Inspection: Extremities, hands and feet, not enlarged. Head large, has increased one or two sizes since he was 25. Eyes prominent, right slightly more so than left. Reflexes normal. Carbohydrate tolerance very high, over 300 grams of glucose. Skin dry and smooth, yellowish not clear, but no brown spots. Thyroids not enlarged.

Vision: Right eye 20/50, left eye 20/40, with correction right eye 20/40 minus 1, left eye 20/20. Ophthalmoscope: About normal, possibly nerve-head a trifle pale in outer quadrant of each eye. Fields much constricted, not bitemporal, relative central scotoma.

Nose: Ethmoiditis on both sides. Operation, resection of anterior tip of both middle turbinates and exenteration of posterior ethmoid cells.

Treatment, sodium salicylate and rest. Noticed slimy discharge from nasopharynx from time to time after the operation. Headaches much better. Vision improved to 20/20 minus 4 in right eye, and fields for white were about normal.

While under treatment an ulcer developed on the left eye which reduced its vision to 20/200. Patient was sent home.

May 11, 1912: Patient returned with vision 20/30 in right eye, 20/200 in left. He still had considerable headache, fields about same; considerable discharge from left ethmoid region.

Aug. 26, 1912: Vision right eye 20/30, left eye 20/50, fields constricted but not so bad as when first seen. Discharge from left ethmoid region and continued headaches. Resected remainder of left middle turbinate and ethmoid cells, and removed anterior wall of left sphenoid.

Sept. 12, 1912: Vision right eye 20/20 minus 1, left eye 20/50, fields for white normal, no headache, no discharge from ethmoids. Slimy discharge from posterior pharynx at times, and head always feels much clearer and better after such a discharge.

Feb. 16, 1913: Vision right eye 20/20 minus 1, left eye 20/70. Ophthalmoscope: Normal, temporal fields about normal.

TABLE 3.—MEASUREMENTS OF FORTY-ONE NORMAL SELLAE TURCICAE*

	A. P. mm.	D. mm.		A. P. mm.	D. mm.
1.	10	8	22.	9	7
2.	13	9	23.	10.5	6
3.	9.5	6	24.	11	8
4.	10	8	25.	10.5	8
5.	10	6	26.	12	8
6.	11	7	27.	11	8
7.	10	8.5	28.	10	8
8.	10	8	29.	12	9
9.	10	8	30.	13	9
10.	10	8	31.	12	8
11.	12	10	32.	8	10.5
12.	10.5	7	33.	11	8
13.	12.5	7	34.	10	8
14.	16	10	35.	11	6
15.	11	8	36.	10	8
16.	10	8	37.	10	6
17.	12	6.5	38.	12	7
18.	11	7	39.	11	9
19.	10	7	40.	10	5
20.	11	8	41.	10	9
21.	10	8			

*Taken from persons examined by Roentgen ray for foreign body or other accident, in which there is no history of headache or other trouble which might point to any disorder of the pituitary gland. This table shows that the great majority of normal sellae turcicae vary within quite definite limits, the average being A. P. 10.8, D. 7.7. In this table No. 14 shows an abnormally large sella. I have no history to show whether the patient had any symptoms of disturbance of the pituitary gland.

This case presents many typical symptoms of pituitary trouble in which there is a deficiency in the posterior lobe secretion. The slimy discharge patient speaks of with the improvement in his vision and cessation of headaches, following the drainage of the sphenoid cells, would seem to point to a probable cystic degeneration of the gland, with drainage through the sphenoid. So long as his vision is not affected and he is not suffering from headaches, I have not advised him to seek an operation, but he understands his condition and I have advised him that if he notices a diminution of his vision or a return of the headaches, an operation will probably be necessary.

The above cases fall in Cushing's classification, Group I, "Cases of dyspituitarism in which not only the signs indicating distortion of neighboring structures but also the symptoms betraying the effects of altered glandular activity are outspoken."

CASE 4.—I present this case to call attention to the changes that may occur in the sella turcica due to pressure from tumors located at a distance.

C. L., aged 20, Dec. 19, 1912, consulted us in regard to loss of vision and headache. Father and mother living; several

2. Since sending in paper I saw patient and carbohydrate tolerance was over 300 gm. glucose.

brothers and sisters, all well. Medium size, appearing younger than he is. Healthy until the present illness.

He first noticed trouble October, 1912, after wrestling match, in which he became very warm. Next day he had severe frontal headache, dizziness, nausea and vomiting; he was sick about a week and then gradually improved. At this time he had pain in right eye and says there was a tendency for his eyes to become crossed. Sight was somewhat blurred. He had pain in head accompanied with dizziness and nausea from time to time, but was not especially sick until about two weeks previous to present visit. At that time ran after cattle and became very much heated; this was followed by vomiting spells and he was unconscious for some hours. There was no constipation, polyuria, or increased appetite for sweets.

Inspection: Medium size, head somewhat larger than normal, hair on face and body scanty, well nourished but not fat. No enlargement of bones of hands or feet, other ductless glands not enlarged.

Vision: Right eye 10/200, left eye 20/40. Ophthalmoscope: Marked papillo-edema in each eye, perhaps a little worse in right eye. Pupils normal, possibly slight exophthalmos, inclined to fall to the right, knee-jerk gone. Fields, homonymous hemianopsia.

Roentgen ray showed walls of sella turcica largely obliterated, and pituitary body much increased in size. A diagnosis of probable pituitary tumor was made, decompression operation suggested, but not accepted. Patient went home.

TABLE 4.—MEASUREMENTS OF TWENTY SELLAE TURCICAE OF HEADS WHICH WERE ROENTGEN-RAYED FOR DIAGNOSTIC PURPOSES IN PEOPLE COMPLAINING OF HEADACHES, NOT INCLUDING THOSE IN TABLE 2*

	A. P. mm.	D. mm.		A. P. mm.	D. mm.
1.	10	10	11.	12	10
2.	12	8	12.	12	8
3.	11	11.5	13.	11	8.5
4.	13	12	14.	12	7
5.	11	8	15.	11	9
6.	12	7	16.	14	10
7.	11	7	17.	12	8
8.	12	9	18.	12	8
9.	13	10	19.	10	6.5
10.	15	9	20.	12	9

*This table varies but little from Table 3. A. P. measurements 11.9, D. 8.7. No. 10 was a case of specific meningitis with no evidence of pituitary disorder. No. 9 was a case of severe temporal headaches, associated with marked diminution of vision (vision 10/200 in each eye); fields for white not restricted, central scotoma, no evidence of pituitary disorder, sphenoid cells very large, Wassermann negative, tuberculin negative. Diagnosis made of probable auto-intoxication. No. 8 is a colored woman. No. 11 has suffered for the past ten or twelve years with very severe bursting headaches. Nos. 1, 2, 5, 16 and 20 have been subject for a number of years to very severe headaches without apparent cause.

When next heard from, February, 1913, he had entirely lost his sight and father was very anxious to have something done. Dr. Gifford advised them to see Dr. Cushing, who made a diagnosis of a tumor of the posterior cerebral lobe on the right side, and accounted for the changes in the pituitary region as probably caused by intracranial pressure. As the boy had been entirely blind for some time and there was practically no hope of restoring his sight, the father again refused operation.

CASE 5.—L. E. B., aged 53, March 22, 1913, complains that sight is poor; he can see straight ahead but not on either side. He was subject to severe headaches, beginning when about 24 or 25 years old, and lasting until five years ago. At that time he had a very serious illness, with severe pain in the head; he became unconscious and remained so for fifty-two hours; was paralyzed on both sides, left side complete and right side partial. He says that both arms and legs were paralyzed; he had to be fed, was blind for several days. He gradually recovered some sight and was able to walk in about two months. Physical condition gradually improved until he felt about as well as ever. He says he has had no headache for last five years.

Inspection: Man of medium height and build; slight drag to left limb. Not fond of sweets; no polyuria or glycosuria. Hirsutics about normal. Right knee-jerk normal, left very slight. July 28, 1910: Vision, 20/20 minus 1 in right eye, 20/30 minus 1 in left eye. Pupil moderately dilated. Ophthalmoscope: Nerves a little pale, fields 20 degrees in diameter.

March 22, 1913: Vision right eye 20/20, and left eye 20/30. Pupillary reflexes a little slow. Ophthalmoscope, nerves somewhat pale, fields as shown in Figure 6. Roentgen ray: Normal outlines of sella entirely obliterated, floor forced down into sphenoid cells. No changes in long bones or phalanges.

The striking points of this case are the fact that the patient had terrific headaches, which nothing would relieve for years, and following an attack of paralysis, during which time he was blind for several days, the headaches have entirely ceased. Central vision returned to almost normal, and with the exception of a slight drag to the left leg, he seems to have recovered entirely from his paresis. Health is good and he is going about his business. There are no indications of pituitary trouble other than the roentgenogram and the eye-findings. If the condition that caused the destruction of the sella was a distal tumor there is no sign of its location at the present time. The question arises if the patient has not had enlargement of the pituitary gland, which, though it has destroyed the normal outlines of the sella, still continues to have a sufficient amount of active gland for the normal functions of the body. I am indebted to Dr. Mick for discovering the disturbance of the sella turcica. When seen by us in 1910, three years after the attack of paralysis, the pituitary body was not suspected.

CASE 6.—Dr. R. D. H., aged 61, examined by my colleague, Dr. Gifford, Nov. 1, 1909. Vision: Right eye, hand movements at 18 inches, left eye light perception. Pupils react to light. Tension normal. Ophthalmoscope: Nerve-heads white. Diagnosis, atrophy. At this time nothing especially abnormal was noticed about patient, except for primary optic atrophy.

TABLE 5.—SKULL AND PITUITARY MEASUREMENTS OF FOUR CHILDREN, WITH THEIR AGES*

	Years	A. P. cm.	D. cm.	A. P. mm.	D. mm.
1.	12	6.25	9.5	7	10
2.	6	5.25	10	8	6
3.	4	5.5	10	7.5	5
4.	15	...	..	10.5	7

*In this table by leaving out the fourth case, aged 15, the measurements are A. P. 7.8, and D. 7. The average of the four is A. P. 8.5, D. 7.

Some months ago Dr. Mick, whom patient interviewed as to the possibility of electrical treatment benefiting the vision, took a roentgenogram of his head, and found the outlines of the sella turcica entirely lacking. Dr. Mick then referred the patient to me for ophthalmic examination.

Vision: At this time, right eye hand movements at 18 inches, left eye, no light perception. Ophthalmoscope: Optic nerves atrophic, and vessels small.

I was not able to ascertain from history more than that he had been subject to headaches at the time his vision began to fail. He is mentally bright except at times he complains of a decreased secretion of urine accompanied by headaches and stupidity. He is of medium height, moderately adipose, abdomen not pendulous; hirsuties normal. I was not able to obtain carbohydrate tolerance; otherwise condition was normal for a man of 65. The amount of adipose could easily be accounted for by his lack of exercise, owing to his defective vision.

CASE 7.—Miss C., aged 40, was seen by my colleague, Dr. Gifford, at an out-of-town sanatorium about Feb. 15, 1913. At this time patient was blind and showed a choked disk in each eye.

March 17, 1913: Patient was brought to office for examination. Condition of eyes was unchanged. She had had severe headaches for some months past, with a rapid loss of sight about February 1, at which time she suffered greatly from headaches and was unable to be around. For past month her physical condition has improved so that she is now able to be about the house. She has no signs of pituitary trouble, except as shown by the Roentgen ray. Thorough examination by a competent neurologist failed to reveal any evidence of other intracranial tumor. Examination by internist was equally fruitless. The roentgenogram showed the pituitary body to be

very much enlarged. The peculiar thing about this plate is that the anterior horns are not disturbed, while the posterior process of the sella is almost entirely obliterated. This probably accounts for the papillo-edema of the optic nerve, which is due to intracranial pressure rather than direct pressure on the nerve by the tumor.

These three cases will fall in Group II of Cushing's, "cases in which the neighborhood manifestations are pronounced but the glandular symptoms are absent or inconspicuous."

To ophthalmologists this should emphasize the importance of having a roentgenogram taken in all cases showing a primary optic atrophy or papilloedema, unless there is some well-established cause for the trouble; as otherwise its true source might not be suspected. They also serve to substantiate Cushing's contention that if these patients can be seen early and an operation done to save their sight they may continue to enjoy comparatively good health for a number of years to come; as by reference to Cases 5 and 6 it will be seen that the loss of the vision dates back several years, and that but for the loss of their sight the patients would be having about normal health to-day. If the enlargement of the gland is not a malignant degeneration, but a hypertrophy or degeneration such as is seen in the thyroid, the pressure symptoms are the only ones causing trouble.

CASE 8.—N. J., aged 13, was referred to us Nov. 21, 1912, by Dr. Anderson for ophthalmic examination, with a diagnosis of polyglandular syndrome as in Group V by Cushing's classification. Patient was undersized for age, markedly adipose, bones small, mentally decidedly below normal, tapering fingers, Fröhlich's type, some evidence of thyroid deficiency, no pigmentation from adrenals. Thyroid gland had been administered for some months previous without much effect. At the time of writing this paper patient had lost 3 pounds after one week's administration of whole pituitary gland. Roentgen ray showed the sella turcica apparently normal, except that it was decidedly large for a child of this age. The anterior-posterior measurement of the sella turcica was 14 mm. and the depth 10 mm. The shadow from the pituitary gland seemed to indicate that it was more dense than normal.

CASE 9.—Mrs. T. B. P., aged 38, came in Nov. 12, 1912, complaining of pain and swelling over right frontal sinus. Father and mother were rather above the average in size, healthy. Patient had had no sickness in childhood or adolescence; had had severe temporal headaches at times for the last eight or ten years; present trouble for about six months. She had perineal operation with removal of portion of right ovary while under observation; it was then found that she had a double uterus, infantile type.

Inspection: Large well-nourished woman, height 5 feet 9 inches, weight 180, no enlargement of hands, feet or head. Skin normal. Hirsuties normal, no enlargement of other ductless glands demonstrable, no increased appetite for sugar, carbohydrate index, or polyuria. Patient never pregnant; menstrual periods regular for the past eight years. Right frontal sinus tender, transillumination right frontal sinus dark; otherwise normal. Roentgen ray: right frontal sinus decidedly blurred, measurements sella turcica, anterior-posterior 15 mm., depth 10 mm. Anterior clinoid processes gone. Vision normal, ophthalmoscope normal, no history of blurring of vision. Pupillary reflexes normal. Operation, resection of anterior tip of right middle turbinate, and probe passed into right frontal sinus. Relief of frontal pain, but some headache still continues.

I present this case as showing a peculiar condition in which, associated with a very severe temporal headache, the anterior clinoid processes are absent. There are no other pronounced symptoms of disturbance of the pituitary body, and yet I cannot but feel that there is a direct relationship between the severe headache, without any other apparent cause, and the enlargement of the pituitary body with loss of the anterior horns of the sella turcica. In the entire series of normal heads which I have reviewed these are the only ones showing the absence of the anterior horns. I shall make a strenuous effort to keep in touch with these cases, as it will be interesting to note if they later develop decided evidence of pituitary disturbance.

In conclusion, an important point to be remembered in connection with the enlargement of this gland is that it should not be classed with the other tumors within the cranial cavity, but with the hyperplasias or degenerations similar to those which we recognize in the thyroid. For while it may undergo malignant degeneration, its enlargement is much more liable to be a hyperplasia, or a cystic degeneration.

Considering it, then, as we would the thyroid, a true gland, we are not surprised that it so frequently becomes enlarged, and were it not for its peculiar location, a variation in its size would be of much less serious consequences. But owing to this we have not only to consider the effects of its deranged glandular activity, but also the effects of pressure on the adjacent structures. Considering it in this light we find it gives us a much more hopeful outlook, for at any time it may cease its enlargement, as has been noted.

Again, by surgical aid we may relieve pressure symptoms and allow for some further enlargement; or should there be a cystic degeneration this may be drained. Then by supplying, if necessary artificially, deficiency of secretion by feeding appropriate doses of the gland, the patient's life may be indefinitely prolonged, his pain relieved, and often the full use of his faculties retained. In order that this may be done it is necessary that these cases be recognized early, else irreparable harm may be done and modern science be robbed of its victory.

ABSTRACT OF DISCUSSION

DR. W. H. MICK, Omaha, Neb.: The largest sella in an otherwise normal man measured 17 mm. anterior-posterior and 11 mm. in depth. So no absolute size can be laid down for the sella or the pituitary body. The size of the sella is, however, more closely confined to certain dimensions than the accessory sinuses or the bony parts generally about the head. When portions of the bony wall are absorbed we may look for a pathologic condition somewhere in the brain, which need not, however, necessarily be located in the pituitary body. A glioma in the brain has nearly the same density that the brain has and may not be easily read on a radiographic plate. Thinning of the skull-walls may not be noticeable early in a very rapidly growing brain tumor. In a number of extreme cases the first evidence of intracranial pressure, recognized radiographically, was a disturbance of the bony outline of the sella. On the other hand, careful radiographic study has shown that infection of the sphenoid and ethmoid cells has caused increased density of the pituitary disease, and that when corrected the density and also the other disturbances, including vision, were improved. It is well worth emphasizing that the clinoid processes forming the anterior roof of the sella are absent in a certain number of cases in which no other pathologic condition is located radiographically. In these cases chronic headache has been one of the complaints. The reason for such headache I cannot give and I trust that someone may be able to enlighten us regarding its cause. Two cases shown with this paper were advanced cases of optic atrophy, the patients having no symptoms of pituitary disease; still the entire sella in both cases was destroyed. This fact led me to investigate changes in early cases of optic atrophy, which were found, without exception, within the limits of normal measurements, etc. The post-mortem examination on Mr. C. L., showing the pituitary body normal and the destruction of the sella complete with an abnormally dense shadow back of it, shows how readily compressed tissues cause an increased density in the Roentgen shadows and even destruction of the walls, which easily explains the pressure on the optic tract. No two of the cases of syphilis in which we

V. 6
1913

found changes in the sella were alike. So radiographically speaking one could not differentiate syphilis infection of the head from changes in the sella. We can, however, suspect syphilis as the cause of these changes in the sella from the fact that the skull-wall is markedly thickened, is irregular in density and, last but not least, by the presence of stellate bodies located in the bony structures.

When changes take place in the pituitary body they are recognized early radiographically, either by a loss of the upper portion of the bony wall or an increase in the size of the sella, or by crowding the sella down in the sphenoidal sinuses. It must not be forgotten, however, that the sella is sometimes found crowded in the sphenoidal sinuses and still normal in dimensions from other causes than pituitary disease. It is needless to mention that in a case of pituitary disease bony changes are recognized elsewhere in the body radiographically. Our study has clearly demonstrated that changes elsewhere in the skull cause changes in the sella turcica and the careful study of Mr. C. L. would convince us that pressure changes destroying the sella, with pressure on the pituitary body may cause an overdevelopment of the maxillary bone and in this case the teeth are usually clear and dense to Roentgen penetration, which is unusual and which I really would not be able to explain in any other way.

DR. JOHN B. POTTS, Omaha: One thing has not been brought out by the different neurologists, and that is that an inflammation of the sphenoid cell may have some effect on the pituitary body as well as on the optic nerve. I have two cases that bear somewhat on this. In one I showed the radiograph. The other is that of a woman who never had any symptoms of pituitary trouble, but had retrobulbar neuritis, during the course of which she developed incipient diabetes, with the passing of an immense amount of water, which has continued even though the nasal and ocular symptoms have cleared up. Dr. Cushing in going over the same thing in operating on dogs found that the increased flow of urine began three days or more after the operations.

SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

PAPERS† AND DISCUSSIONS

THE DIGNITY OF OTOLARYNGOLOGY *

ROBERT LEVY, M.D.

DENVER

The department of otolaryngology represents one of the refinements of medical science and as such should be bounded by no narrowness of view or bigotry of opinion. Its members should be recognized as representing the highest ideals. Its officers should be chosen not by political intrigue or for the purpose of self-aggrandizement, but because of their actual worth and honorable conduct and their recognized ability. No other consideration has a place in a scientific body such as this. Political faith or religious creed become subservient to and are forgotten in the enthusiasm for scientific attainment. Believing this and recognizing in my predecessors men whose names stand for the noblest ideals and for the greatest scientific value in our specialty, I am profound in my appreciation of your action in placing me in the same "Hall of Fame" with such associates. No man can be unmindful of this commendation at the hands of his colleagues, and so I wish in these poorly chosen words to extend my sincere thanks.

The love and respect in which we all hold our specialty has been the motive that has given rise to the thought expressed in the title of this address and, as Stevenson puts it, "there is no subject of which a man should speak so gravely as that industry, whatever it may be, which is the occupation or delight of his life; which is his tool to earn or serve with; and which, if it be unworthy, stamps himself as a mere incubus of dumb and greedy bowels on the shoulders of humanity."

The history of otolaryngology is intimately interwoven with that of the American Laryngological Association, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Laryngology and this, the Section on Laryngology, Otology and Rhinology of the American Medical Association. In the early days of this specialty laryngology played the most important part. Its adherents were big men, who as pioneers exerted their utmost to place this branch of medicine on a high and dignified plane. That they succeeded in launching this department on a successful career has never been questioned, notwithstanding the criticism and ridicule with which the laryngoscope, a so-called toy, was received by the general profession. One has but to read the address of Louis Elsberg at the first meeting of the American Laryngological Association to appreciate the ambitious future planned for that society which, in a measure, reflected the hopes of those interested in this specialty.

Among other things it was the wish to develop a proper nomenclature for the diseases with which we deal and to increase the importance of our specialty by the establishment of chairs of laryngology in the various medical schools. While much has been done toward clarifying the nomenclature used it cannot be said that this is as yet by any means perfect. The importance of our branch of medicine has been greatly enhanced, however, by the fulfilment of Elsberg's desire for more general instruction in medical colleges.

The entire trend of the addresses of early writers is in line with the loftiest possibilities. And while it is admitted that the special societies, special journals, and individual workers have in a measure more or less satisfactorily fulfilled the ambitious hopes of our pioneers and have advanced toward that goal at which our specialty would be raised to the highest and most dignified position possible, nevertheless it must be confessed that there creeps in from time to time much that is degrading and that detracts from its best achievements.

In the early days laryngology, as such, occupied a most exalted position and its votaries were men of the highest type. Rhinology was at that time but slightly in evidence; in fact, from 1855 to 1878 in a total of 1,766 papers collected by Elsberg, less than 7 per cent. dealt with diseases of the nose. As the specialty grew in point of numbers, rhinology, as such, attracted widespread attention. Coincident with the growth of rhinology there appears to have been a depreciation in the tone of the specialty. This must be largely attributed to an unscientific era and the beginning of an age of commercialism. "Catarrh" specialists became numerous and the unscientific management of diseases of the nose, not to mention the advent of charlatanism in the treatment of these conditions, was perhaps the severest blow to the dignity of our specialty that it could possibly have received. Men entered the profession, and in certain instances are entering it to-day, with the belief that all that is needed to become a throat and nose specialist is a spray apparatus and an unblushing nerve for charging high or so-called specialists' fees. Many a case of deflected septum was treated by local applications, the temporary improvement being the result of a temporary reduction in the turbinate engorgement. Fortunately a halt to this state of affairs was called when surgery of the nose and accessory sinuses became the recognized field of the rhinologist. Later otology still further raised the standard and added to the dignity of our department and, although the early otologist was nearly as empirical as the early rhinologist, it is gratifying to record that no department of medicine or surgery has developed more rapidly or with greater or more accurate and intricate scientific advancement than that dealing with the organ of hearing.

Beginning then with laryngology and its high ideals carried out in practice, to which was later added the debasing influence of early rhinology, and again approaching the noblest principles of our profession by the addition of advanced otology, what is needed to continue toward that proud and dignified position which we all trust may be ours?

First and foremost we should demand a high moral tone of every man who enters our profession. The material tendency of the times has to a certain extent obscured the necessity of making our practical lives virtuous and righteous. The man who enters the profession of medicine with the proper appreciation of the

† As the footnotes indicate, certain papers are here printed in abbreviated form.

* Chairman's address.

V. 6
1913

nobility of his calling, however, will be led naturally to avoid the degenerating influences which so easily creep in. For men endowed with the proper sense of their responsibility and with due regard for the traditions and ideals of our profession it becomes a comparatively easy matter to disregard and to eliminate the commercial spirit of the age. The greed for gain becomes a vice and fee-splitting and commissions become abhorrent.

In addition to a strong underlying nobility of character it becomes necessary to fortify oneself with the proper preparation. Mere sentiment cannot bring about any sort of practical results. The standard of premedical education has been gradually raised and in no department is it more necessary to possess the highest scientific and literary attainments than in our specialty. The regular four-year medical course and hospital training are also only preparatory to the development of a competent specialist. In this connection it is well to call attention to the excellent report of the committee of the American Laryngological, Rhinological and Otological Society which was appointed to consider the best methods to be followed in the teaching of otolaryngology. Other societies will act in conjunction with this committee. The report, which has not been published as yet, outlines a tentative plan of study which must of necessity improve the quality of the men entering our specialty. It condemns, as we all should, the making of an otolaryngologist by a six weeks' postgraduate course and, as the report states, "it is time we had done with this farcical sort of preparation, if our specialty, worthy as it is of the best, is not to be dragged in the mire, as a result of the ignorance of anatomy, diagnosis and technic, displayed by a very large proportion of the rank and file of those who now style themselves specialists in diseases of the ear, nose and throat."

Lastly, much can be done in the every-day practice of our profession toward dignifying its position as a department of medicine and surgery. The slipshod, careless and unscientific management of many of our operative procedures have placed them in a class by themselves frequently looked on by the general profession as evidence of a trivial specialty. Even though patients seldom die as the result of our operative interference, even though Nature covers up the possible serious results of careless ear or nose or throat operations, even though perfect asepsis cannot be attained in every operation owing to the peculiarity of the field attacked, nevertheless every procedure should be treated with all the refinements of modern surgery. Just as we are making tonsillectomy a hospital and not an office procedure, just so should we treat the majority of all intranasal operations. The management of the middle turbinate is no longer confined to this structure but includes an attack on the entire ethmoid. Septum operations and intranasal approach to the accessory sinuses may be followed by dangerous consequences. The shock of many a so-called minor procedure, such as incision of the membrana tympani, has been known to have serious after-effects. The welfare of our patients, the avoidance of unfavorable complications and results, and the dignity of our calling demand that in operative interference wherever possible the office should give way to the hospital. The upright position should give way to the recumbent. Careful preparation of the patient, the cleansing of the face, covering it with a sterile sheet for intranasal work as in ear work and, in fact, every known method of surgical cleanliness should be insisted on.

These ideal, as well as practical, considerations become a compelling force which will increase our own self-respect, advance the standard of our specialty and raise it to the dignified position which its importance in medical science demands.

Metropolitan Building.

CONSERVATIVE SURGERY OF THE NASAL SEPTUM

IRVING WILSON VOORHEES, M.S., M.D.
NEW YORK

There is probably no operation in the entire field of otorhinolaryngology which is more frequently performed than submucous resection of the nasal septum. It seems wise, therefore, at this time to consider the reasons for operative attack on this structure.

In the first place, we must remember that the septum is in many cases a minor cause of nasal insufficiency. Such a thing as a straight septum is very seldom found, for the reason that Nature never builds any of her structures on absolutely mathematical lines. She is fond of turnings and windings, of curves and spirals, and usually travels by an indirect path to the goal which she has in mind.

Many of us have been surprised to find what we considered a badly deformed nasal septum in a patient having a good intake of air. In these cases there is often no difficulty in breathing, a fact which seems impossible from the examination. Before undertaking operative procedures in the nose, we need to make, therefore, a very careful diagnosis not only of the middle wall (septum) but of the external walls and of the accessory sinuses as well.

The causes producing nasal insufficiency may be far anterior, far posterior, or between these two extremes. Those far anterior in the vestibule of the nose are due to congenital or acquired deformity. One special type needs a word of explanation. We occasionally see patients in whom it is difficult to find a cause consonant with the symptoms of which they complain. On forcible inspiration, however, we find that there is a sinking in of the alae nasi, a valve-like action, which absolutely cuts off the air from entering the anterior nasal orifices. This is due to a weakening or insufficient development of the accessory muscles or cartilages in the wings of the nose; consequently on forced inspiration the strong negative pressure within the nose combined with the external atmospheric pressure produces a marked degree of occlusion.

The turbinates are also frequently responsible for nasal obstruction. This is due either to bony hypertrophy or to hypertrophy of the soft parts. Bony hypertrophy is relatively rare and occurs chiefly in the middle turbinate as a so-called "cystic" condition which in reality is simply an extruded ethmoid cell placed in the middle turbinal bone. Soft hypertrophy is either of local or constitutional origin. The local causes are vasomotor rhinitis, hyperplasia, polypoid degeneration, and swelling of the erectile tissue due to the inhalation of dust, irritating fumes, etc. The chief constitutional cause is chronic passive congestion due to intestinal stasis or to some heart or kidney condition.

Beside these causes, nasal discharge is a very important element in bringing about nasal insufficiency. This arises either in a sinus or comes from the mucous membrane. It may be acute or chronic. The acute occurs

in rhinitis and sinusitis and in infectious diseases such as diphtheria and influenza. The causes of chronic discharge are either local or constitutional, and may be found in association with a bad heart, lungs or kidneys, a chronic gastro-intestinal disorder, or, in short, any disease which alters the quantity and quality of the normal nasal secretion.

In chronic sinusitis one of the important symptoms is the discharge of a thick purulent or mucopurulent, viscid, tenacious fluid which falls down into the nasal fossae, dries and forms firm, tough crusts. These may absolutely occlude the lumen of the nose and are often quite difficult for the patient to remove. Patients thus annoyed are frequently troubled with epistaxis because in their efforts to remove the accumulated crusts with the finger, hair pin, etc., they injure the nasal mucous membrane and bleeding is very easily induced when the nose is blown. If, therefore, a simple nasal deflection exists in such a case it does very little good to remove the septum since the discharge persists and therefore the obstruction also. Here the primary cause of nasal insufficiency is not bony obstruction, but what we may properly term "catarrhal obstruction." I have seen such conditions in patients having a chronic appendicitis and fermentation and putrefaction in the gastro-intestinal canal. There was marked improvement in the quality and quantity of the nasal secretion and in the breathing following appropriate internal treatment.

In the nasopharynx the chief causes of obstruction are hypertrophied posterior-inferior tips, adenoids (ends of the turbinates), postnasal discharge and choanal polypi. Posterior tips (hypertrophied ends of the turbinate) are an expression of chronic passive congestion, and are practically always an extension of inferior border hypertrophy of the lower turbinate. Adenoids in the adult are usually undergoing fibrous contraction and therefore only infrequently obstruct the choanae. Postnasal discharge not due to hypersecretion from the intranasal mucous membrane comes from sinus disease.

All the above-mentioned facts are well known, and my only reason for referring to them here is to present a background for this essay. I am confident that inexperienced and careless rhinologists are in the habit of ascribing every variety of nasal insufficiency to a deviated septum. This is unscientific and thoroughly reprehensible; for it is sure to bring an excellent operation, that of a submucous resection, into ultimate disrepute. There are at the present time too many *middle-line* operators—men whose surgical endeavors are limited to the septum, adenoids and tonsils.

The septal causes of nasal obstruction are deviations, thickening of bone or cartilage, old fractures and dislocations of the integral parts of the septum, spurs, ridges, gumma, abscess and the so-called tuberculum septi. For each and every one of these the submucous resection has been attempted by youthful enthusiasts. Needless to say, the diagnosis of the cause of the nasal insufficiency has frequently proved faulty. I have seen abscess and gumma operated on by the inexperienced who thought the case one of septal deviation.

What harmful or annoying results may follow extensive removal of the septal framework? These are, in brief, perforations, diminished resonance, particularly in singers, weakening of the external nose, subjecting it to deformity from subsequent injury, saddle-deformity, fracture of the cribriform plate with resultant meningitis, destruction of the anterior palatine vessels and nerves with attendant neuralgias and anesthesias of the upper air-ways probably due to deficient blood-supply, and the "flapper" effects. I am well aware that some, at least, of these results are infrequent; and, considering how constantly this operation is being performed by all sorts of men from orthopedists to pediatrists, it is surprising that bad results are not even more common. There is, however, a growing tendency on the part of many of us to perform a far too radical resection. Some surgeon-rhinologists are not content unless they have removed everything high up, far back and along the floor, doubtless because they have been unsuccessful in curing previous cases by a less radical operation. They have failed, however, to take into account the persisting "catarrh" which was probably the original cause of the insufficiency.

Now, the purpose of the submucous operation is to straighten, not to remove, a crooked septum. The bone and cartilage are normal, there is no disease of these structures requiring amputation. The septum is simply misplaced or redundant, that is, there is more solid framework than can adapt itself to the vertical plane. Therefore, why remove *all* of this framework? Obviously all that need be done is to take out the redundancy and fracture the crooked elements so that they may be held in place by simple splinting. Naturally enough, any great thickening of cartilage or bone or any angle which holds the flaps away from the middle line should be removed. These belong, however, to the rarer complications.

OPERATION

The procedure which I have been performing for the past year is in reality a submucous Asch operation. The same care is taken with the elevation and separation of the soft parts as is taken in the radical submucous method. Redundant curves and angles which cannot be reduced and held to the middle line are exsected. The remaining fragments are then fractured and placed in the vertical plane. This can be done with the instruments of the Asch outfit, or with the set devised by Roe of Rochester. When the deviation is limited to the ethmoid, I find it very helpful to go into the nostril with the little finger wet with mercuric chlorid solution or anointed with sterile petrolatum. It is important that the finger-nail be trimmed very close in order to avoid wounding the mucous membrane. The perpendicular plate is then fractured by pressing firmly against it. This bone is often quite thin, and yields readily to this maneuver. Any simple method of packing can now be used. I prefer long strips of sterile gauze saturated with liquid petrolatum. The packing is begun high up and far back and gradually piled up in layers down to the floor. This prevents postoperative hematomas between the flaps and external hemorrhage as well. The packing is simply laid in snugly, not jammed in. In twenty-four hours it is removed. After-care is limited to the prevention and treatment of crust formations in the nose. The patient is cautioned to leave his nose alone, and is not even allowed to blow it for the first three or four days.

SUMMARY

The following simple statements may be used as a guide in conservative work on the septum:

Indications for Resection.—1. When treatment of an associated local or constitutional cause of nasal insufficiency does not give adequate relief of the symptoms.

2. When the deviation is probably the chief cause of obstructed nasal breathing or "drainage."

3. To afford more room when operating on the sinuses by the intranasal method.

V. 6
1913

4. Preliminary to operation on the hypophysis.

5. Preliminary to Yankauer's operation on the lacrimal duct.

Rules.—1. Before attacking the septum, study the outer walls of both nasal chambers at two or three sittings. Adrenalin (epinephrin) and a probe are indispensable.

2. Remove *only enough redundant* deviation to make room for fixation of the remaining fragments in the same vertical plane.

3. Put the septum in the middle line and hold it there.

14 Central Park West.

COMPLICATIONS THAT MAY ARISE DURING OR AFTER OPERATION FOR CORRECTION OF SEPTAL DEVIATION

G. W. MACKENZIE, M.D.
PHILADELPHIA

COCAIN OR EPINEPHRIN POISONING

With cocain the patient develops a paleness of the face, trembling of the extremities, and when most pronounced the patient faints; at the same time the pupils dilate slightly. Alcoholics develop loquacity and boastfulness, combined with fear. I have never observed any poisoning effect from cocain that I could truly classify as alarming. I find that if the patient is placed in a prone position and reassured the symptoms pass over promptly. Amyl nitrate is perhaps the best physiologic antidote.

Epinephrin produces a set of symptoms entirely different from those of cocain. The patient complains of a throbbing sensation in the whole body, but particularly in the head and chest. The face becomes cyanotic, the pulse rapid and of high tension. These symptoms pass over promptly as a rule.

Air embolism may occur from the injection of Schleich's solution, and I have seen it on two or three occasions when a defective syringe was used (the piston fitted too loosely in the barrel).

SURGICAL COMPLICATIONS

Incomplete and improper primary incision of the mucous membrane may be a cause for delay in the first steps of the operation. I have seen beginners delayed in their efforts at separation of the perichondrium through failure to observe this precaution; at the same time they lacerated the mucous membrane considerably.

Adhesions.—So-called adhesions may be a cause for a delay in separating the mucoperichondrium farther, and in a measure may be responsible for a perforation of the mucous membrane. Many of these so-called adhesions do not exactly exist as such; they are found, however, occasionally where there has been a previous fracture. The best way to prevent this embarrassment is thorough filtration of the mucous membrane before beginning the operation.

Old Fracture.—Pre-existing fracture of the cartilage with overlapping edges may be a factor which is responsible for a perforation (with the raspatory) of the mucous membrane of the opposite side. Proper infiltration as outlined in my description of technic, together with precaution against using undue force, is the best means of avoiding perforation of the mucous membrane.

Perforation.—Unintentional perforation of the mucous membrane during the attempt at separation of the mucous membrane may be unavoidable occasionally. Such perforations are not so serious when recognized as when unrecognized, for when it is unrecognized the operator is likely to enlarge the perforation still more. The causes of perforation are imperfect infiltration of the mucous membrane, so-called adhesions, fractures with overlapping edges, recklessness and lack of experience. One or two buttonholes in the mucous membranes need cause no anxiety, providing two of them do not occur at corresponding points of the two sides; then they are liable to be followed by a through-and-through perforation. A successful means that I have found to replace the flaps, prior to applying the dressing, is to have the patient blow the nose forcefully, first one side, then the other.

The breaking off of a piece of the chisel in thick bone may rarely occur in those cases in which there is a low broad spur. To avoid it, I find it safer to remove smaller pieces at a time, rather than to attempt the removal of a larger piece.

Hemorrhage.—Excessive bleeding during the operation may be a complication of sufficient importance to cause alarm, and prompt some rhinologists to abandon further efforts at the operation. Excessive bleedings are usually venous, and occur during operation low down and in front; therefore, it is my practice to attack this quarter after all other parts have been taken care of. Secondary bleeding I have never witnessed and cannot speak of from personal experience.

Fracture.—Unintentional fracture of the septum near the posterior extremity may, in spite of otherwise excellent work, prevent the operator from obtaining as satisfactory results as had been anticipated. A faulty position of a fracture of this kind may be re-set into a more favorable position some days after the operation.

Packing.—Faulty packing of the nose may cause a curling of the edge of a torn flap in on itself, so that the mucous surface of the flap may lay in direct contact with the raw surface of the mucous membrane of the opposite side. This results in undue reaction and thickening, and delays recovery.

Faulty packing may result in getting a piece of gauze through a buttonhole and between the layers of the mucous membrane of the two sides and thus prevent prompt healing. Furthermore, it occasionally happens, as a result of faulty packing, that a piece of the gauze may work loose, posteriorly (aided by hawking of the patients), and dangle down into the pharynx, and even so far as to touch the epiglottis, causing gagging and fits of coughing. It is my practice in packing the nose to use short strips of one-inch gauze, so that in the event of a piece working loose posteriorly, it can be removed without disturbing the whole dressing.

Infection.—A mild infection of the wound may occasionally occur, especially where a pack is used. I have seen two cases, of my own, where the infection was quite severe. These severe infections showed intense swelling and redness of the whole membrane, accompanied with fairly profuse, thin discharge, the whole condition lasting two or three weeks. These cases showed at no time any apparent affection of the accessory sinuses; accessory sinus disease, however, together with the pack in the nose, probably is as frequent a cause of infection of the septum, after the operation as any other. One of the causes of severe reaction after the operation I have found to be the combining of the septum operation with one or more operations on the turbinates or accessory sinuses. Diseased tonsils or adenoids are not to be forgotten as possible causes for infection after the septum operation.

Flattening of the Nose.—Depression or flattening of the nose occurs rarely after the septum operation. The two most important factors leading to this deformity are a too liberal removal of the cartilage anteriorly and infections following the operation.

Hematomas.—These usually occur high up and anteriorly. It is manifested by a bilateral circumscribed swelling of the septum. It may be prevented, more or less, by proper packing of the septum as outlined elsewhere.

Erysipelas.—This is a complication about as prone to follow the septum operation as any other operation. I saw one such case while doing postgraduate work. This case, I recall, occurred in a patient who, after his operation, sat and conversed with another patient affected with erysipelas, in the waiting-room of a clinic. I never learned what happened to this patient eventually, for he was transferred to a hospital for infectious diseases.

Empyema.—Acute empyema of one or more of the accessory sinuses is a complication that may follow in the wake of the septum operation. Some of the cases suspected as being acute empyema may have been nothing more than acute recurrences of unrecognized chronic empyema. It is well, therefore, in suspected cases of empyema, to attempt its cure before operating on the septum, unless there are vital indications to the contrary.

1831 Chestnut Street.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. VOORHEES AND MACKENZIE

DR. C. F. WELTY, San Francisco: Prior to operating on patients in the recumbent position, I had considerable difficulty in the fact that my patients would become faint. Since adopting the recumbent position for my patients, I have had some unfortunate experiences, but none so bad as when I operated on patients in the sitting position. I feel confident that the danger of cases of so-called cocainism is lessened when one operates with the patient in the recumbent position. However, I do not believe that these conditions are due to cocain poisoning at all, but to fright and apprehension. Patients suffer more from the effects of the epinephrin than from the influence of the cocain.

DR. R. BISHOP CANFIELD, Ann Arbor, Mich.: I think one point is very important, namely, that a careful physical examination should be made of all patients who complain of insufficient nasal breathing. After making a careful examination of patients on whom I intended to operate I have been surprised to find many of them afflicted with heart and kidney lesions. The shortness of breath is frequently the result of heart or kidney disease, and the exact condition of these organs must be taken into account before making a prognosis as to the result to be secured by removing the nasal obstruction.

DR. WILLIAM E. CASSELBERRY, Chicago: Although sympathizing, in part, with the conservative attitude of Dr. Voorhees, I feel that he overdoes conservatism. He asks why one should remove any straight portion of bone or cartilage from the nasal septum. The reason involves a fundamental principle of the submucous operation, that the forward part, even though straight, a condition which is rare, is to be removed to give proper submucous access to the crooked part behind it.

While it is true that systemic conditions may alter the quality and quantity of the secretion and otherwise contribute to production of nasal obstruction, yet in this respect our knowledge is so vague and relief by systemic treatment alone is so uncertain that to attain a definite cure within a reasonable period I have found it necessary to remove the obstruction surgically, afterward seeking to guard against recurrence by remedying, so far as possible, underlying systemic faults. Structural deformities and tissue overgrowths, when resulting from faulty metabolism, have usually been years in developing and their retrocession through systemic treatment, if possible at all, is equally slow. Not that complete submucous resection of the septum is invariably indicated, for I think all have occasionally found a limited modification to meet the requirements.

There are, I believe, three or more classes of cases in which one is justified in adopting a partial submucous operation. In the high thin Roman nose I have hesitated, even in adults, to remove such an expanse of bone extending forward to the tip of the nose and upward to the nasal dome, lest the lateral resistance of the external nose be impaired. By a modified submucous resection, sometimes unilateral, but often supplemented, done then or later, and by submucous removal of inequalities from the opposite nostril, I have secured, not a perfectly straight septum, which is rare by any operation, but a septum straight enough for practical purposes.

In a second class there is a moderate deflection, very slight at the front, but gaining volume toward the rear of the nostril and surmounted far back by a bayonet or rosette-like spur of very hard bone projecting across the nostril and even pressing into its opposite wall. The harm in this case is mostly by pressure irritation and obstruction from the spur component which, if closely removed, after loosening of the mucoperichondrium on one or both sides and if the weakened septum perhaps is then simply pressed toward the center line, leaves adequate nasal space. If, however, one judges the deflection component to be bad enough to necessitate the subsequent use of a splint one would instead better make the complete submucous operation. The prolonged use of a splint in connection with a removal of the Asch and Watson-Gleason methods, I would consider very objectionable.

The third class includes children under 14 or 15 years of age, in whom all agree that the complete operation is contraindicated.

DR. HENRY BASSETT LEMERE, Omaha, Neb.: In many cases in which patients appear for submucous operation the erosion on the portion of the convexity presented to the anterior nares has reached the extent of absorbing the entire thickness of the cartilage within the area of ulceration. If this is the case and the ulcerated area is healed before the operator sees the patient, he may find the location of his primary incision to have no septal cartilage. This has happened twice in my experience and I see no positive way to forestall the dilemma, as the resiliency of the septum is not markedly affected by this small absence of its cartilage. By making an incision further back I was able to avoid a permanent perforation. In packing I use a self-packer and a one-inch gauze bandage packing into a finger-cot. Dr. Casselberry is to be given credit for this method. I have found it cleanly and humane.

DR. WILLIAM L. BALLENGER, Chicago: I agree with what Dr. Voorhees said, although he has a round-about way of explaining his work, and I think that it is somewhat far-fetched. In most cases we find obstructions due to some involvement of the ethmoidal sinus, a catarrhal ethmoiditis. This results in a watery discharge in the nose, which irritates the turbinate body and causes a turgescence. Such a condition is usually ascribed to some anterior obstruction in the nose. Again, I have found that a high deviation of the septum, or of the perpendicular plate, is more susceptible to improvement by operative measures than a low deviation. In other words, a large spur in the nose can be attended to with good results and with but little discomfort to the patient.

I think that Dr. MacKenzie is mistaken in saying that he is dealing with adhesions; I believe that it is normal tissue. It was Dr. Neuman of Vienna, I believe, who claimed credit for this operation. Dr. MacKenzie described a very difficult method of elevating. If you adopt his method you will have the same difficulties that he now has. The point of least resistance is directly under the bridge of the nose and here you can introduce the instrument under the cribriform plate and elevate the whole, but only part of the way; on the other side, you can elevate it the whole distance.

V. 6
1913

DR. H. W. LOEB, St. Louis: We should make this a hospital operation and do it as nearly aseptically as possible. This work should not be attempted in the office. Again, this work is best done with the patient in the semirecumbent position. The operator can see better and the position is less fatiguing. There is less danger of cocain poisoning when the patient is in the recumbent position. The method of administering anesthesia is, after all, a personal equation. I cocainize the parts and follow with epinephrin. I have seen no bad effects from the use of epinephrin and cocain in this way.

Dr. MacKenzie spoke of the fibers observed when making the incision; if one does not cut entirely through the perichondrium he is apt to be confused by these fibers. The white, glistening cartilage should be noted before one starts to elevate.

I have been through all the stages of packing and I have found satisfactory results from packing removed within six or eight hours. I have had to repack only once in five years. I always have in mind the prevention of septicemia, in view of the fatal case that occurred under the care of Dr. Harold Hays of New York.

I disregard almost entirely the occurrence of hematoma. I have seen it occur, especially when the packing has been left in over night, but I have always found that after a short time it goes down. I have never had to make an incision for hematoma or abscess and I am not at all worried about these conditions.

DR. EMIL MAYER, New York: I wish to endorse the statements made by those who favor the recumbent position in these operations. I have never done a resection operation with the patient sitting up. Those who have been in the habit of operating on patients in the sitting position should try operating with the patient lying down. It may be a little more trying on the doctor, but it is certainly more comfortable for the patient. Patients operated on in the sitting position often topple over, and the operation has to be stopped until they regain their balance.

My method of using cocain applications is to apply a 10 per cent. solution for ten minutes by the watch; then a solution of epinephrin ten minutes by the watch. Then I can operate and have no trouble whatever. Occasionally, however, one meets the experience that I had the other day. The patient gave me some reason to believe that she had cardiac disturbances. I operated using the solutions of alypin and epinephrin, applying them to the mucous membrane ten minutes by the watch, and I had no trouble whatever.

The use of the rubber covering is at times a valuable procedure, but I have what I believe to be a simpler one which answers the purpose. I use a small piece of rubber tissue of two or three thicknesses and about one-half an inch in width. This I carefully introduce into the side of the nose, leaving a portion lying outside. I can pack against this all I like. It is my suggestion not to operate on young children; I think that all will agree with me in this.

The whole gist of the suggestions made in both papers is, be sure you are right and then go ahead.

DR. GEORGE PAULL MARQUIS, Chicago: One condition has given me much trouble, the symptoms of collapse. I have been in the habit of blaming the cocain for these symptoms. I have used the Schleich's anesthesia, the infiltration method, but have not used epinephrin in solution for some time because of a disagreeable experience. I have not had any trouble at all in my operations except from the hemorrhage which occurs after I chisel off the base. I have a comparatively bloodless field up to that time. I have been using peroxid of hydrogen for some time and with its use I have been better able to control the hemorrhage than when I used epinephrin.

DR. CORYDON G. DWIGHT, Madison, Wis.: Dr. Voorhees seems to have reverted to the old method of submucous resection. There is a point at which the middle turbinate comes in contact with the septum. This point should be kept in mind in these submucous operations. I think that the recumbent position in operating is advised by most operators. I have devised a table which can be elevated eleven inches; the patient can be placed on it in the complete or the semirecumbent position. An assistant controls the head by raising or lowering the table. The sides of the table are hollowed out, permitting the surgeon to stand in front of the patient.

I believe that the epinephrin solution should always be made from the fresh tablet sterilized.

DR. J. HOLINGER, Chicago: Dr. MacKenzie spoke of doing the submucous resection and afterward at a second sitting, other operations necessary in order to give free breathing space in the nose. I should like to hear Dr. MacKenzie give his reasons for not doing everything at once. In my experience the cases are not infrequent in which a deviated septum is combined with hypertrophic turbinals, especially lower turbinals. If you do not correct both the septum and the turbinals you have not got the patient in a position to breathe through his nose. But why do you make two operations of it? I believe that we should do the entire operation at once. If there is a hypertrophy of a lower turbinal, remove it, and, if necessary, do the same thing on the other side. We shall often find swellings that are not confined to the nose alone, but which extend to the nasopharynx as well. In such cases you will get no results at all from any single operation. If the nose is properly cocainized, a complete operation will not, in my opinion, complicate matters.

DR. S. R. BOYCE, Madison, Wis.: In my experience the work on the middle turbinate described by Dr. Holinger corresponds to what should be done in cases with the so-called sigmoid deflection; therefore, I nearly always do a partial trimming of the middle turbinate in the larger side. If the inferior turbinate fills in the opposite side, it should be reduced to the normal size at the same time the septum is resected.

I should like to bring out in regard to the posterior ridge, which is often so marked and so far back. If one examines the nose with the posterior rhinoscope, he may not see this because the posterior end of the vomer is nearly always symmetrically placed. In these cases the triangular cartilage almost always overrides and comes down beside the septal ridge of the maxillary bone, so that in resecting the cartilage there is a long strip by the side of the maxillary ridge which might be missed. This I have found in many cases, and it usually terminates in a mass of bone and cartilage on one side about 2 cm. anterior to the posterior border of the septum.

DR. FRANCIS EMERSON, Boston: There are several indications for operation, such as pressure symptoms, deficient drainage and impaired oxygenation. There is one type of case, the one with the high deviation of the septum, which is particularly obstructive when the patient lies down. These cases have a bad effect on the general condition, with restlessness and the disturbance of sleep from a diminished supply of oxygen. Whether one should do the partial or the complete resection operation depends on what has happened in the nose previous to the time of operation. If there is a deflected septum which interferes with respiration, causing irritation symptoms, there will be present an increased mucous secretion and a perverted or over-function from the increased irritation or stimulus, resulting in a catarrhal condition of the mucosa. If this process goes on, the mucosa becomes thickened and the ostia blocked; the end of the middle turbinate also becomes thickened, or the seat of a hyperplasia, and it may be necessary to remove enough of the middle turbinate so that an operation on the septum in order to relieve the respiratory trouble may not be necessary. I think that in considering septal operations, these secondary effects should always be kept in mind; if drainage is interfered with from secondary hyperplasia, there is the possibility that the relief which is expected from a septal resection will not be obtained.

DR. SAMUEL G. HIGGINS, Milwaukee, Wis.: The method suggested consists, in fact, in an expansion of the upper maxillary. It should be borne in mind that children and young adults with badly deflected septums suffer from malnutrition, and in these patients we should improve the condition of the teeth and increase the size of the nasal spaces, correcting deviations, etc.

DR. HOWARD F. PYFER, Norristown, Pa.: The Asch operation has its place, but it should not be used in combination with

the submucous resection. To injure the delicate mucous membrane with Asch's forceps invites a perforation from sloughing, if not from the mutilation.

Dr. Freer's flap method of resection may be used by some successfully, but in my hands it was a failure. The dangers of fracturing the cribriform plate, so much feared by Drs. Voorhees and MacKenzie, can be avoided entirely if, after the removal of the cartilage, the bony septum is bitten out above the ridge. This leaves a gap between the base and the horizontal plate of the ethmoid and thus prevents the transmission of any force used in breaking the bone with forceps or in chiseling the base. If a preliminary hypodermic of morphin is given before the use of cocain and if the cocain is dissolved in a 1:3,000 epinephrin solution, no constitutional effects and no operative shock will be experienced.

I believe that many perforations are due to faulty technic and blundering on the part of the operator, and not to atrophic membranes or adhesions. If an operator is having perforations (as I sometimes did when I used methods now advocated by Freer and Voorhees), he should abandon his technic and learn another and more exact one. Then he will cease making holes.

If gauze packing is used, first place rubber tissue next to the mucous membrane. There will then be no bleeding or injury to the membrane when the dressing is removed.

DR. J. C. BECK, Chicago: Surgical pathology teaches that, after fractures, bone originates from bone, especially when it has periosteum attached to it. How one can prevent new bone formation after fracture I do not know. This is a point which I think should be borne in mind. I consider epinephrin a dangerous drug. I do not think it wise to use it.

I think that the position is a matter of choice. The recumbent position is better for the patient, but the operator can do quicker work with the patient in the sitting position. I recall one case, in which a patient was being operated on in the sitting position; he suddenly moved his head and a perforation resulted. This would not have happened if he had been in the recumbent posture.

DR. JAMES J. PATTEE, Pueblo, Colo.: It is generally stated that the recumbent position is the proper position in doing septal work. Five or six years ago I was doing some work on the septum with the patient in the upright position. After he had been cocainized, he became faint. He was very nervous and I punched right through the vertical plate of the ethmoid. The injury was irreparable. These accidents will happen with the patient in the sitting position. If he is in the recumbent position they will not occur. I therefore recommend that patients be placed in the recumbent position and also that they be given morphin before the operation

DR. G. SLUDER, St. Louis: One complication has not been mentioned, the subsequent sloughing of the mucous membrane. I have seen this occur about five times. If the membrane is lifted and the septum straightened out, overcoming the deformity, you will find that for a week or ten days following the operation the patient will be all right. Then a perforation will appear. I had a case of this kind about six months ago. The patient, a man aged 20, had a perforation that was supposed to be disastrous. If any rhinologist saw it later, with or without asking particulars about it, he might rise up against me. I asked this young man to return to me in about six months. He did, and to my amazement and disgust he showed a second perforation in the posterior half; this second perforation was larger than the first and was a forward perforation.

The point raised by Dr. Beck is a timely one; I do not feel guilty when I make a perforation of this kind. I have had another case in which the opening appeared ten days after the operation. In this case I did a limited resection; in the first case I did a more extensive operation. I do not believe that we are justified in stating that we should not have these perforations and that if they do occur the operator is to blame.

There is no doubt that disaster will sometimes follow the use of epinephrin, but I get along with the least possible amount of the drug and have no trouble.

DR. J. A. STUCKY, Lexington, Ky.: First I wish to place myself on record as advocating a greater harmony among the otorhinologists and the general surgeons in their hospital work. Secondly, I believe that in this operation the patients should be placed in the semirecumbent position.

Nothing has been said about prevention of the toxemia from the use of cocain. I should not dare to use the same amount of cocain where I live as is used in the East and the West. The people of the South are peculiarly susceptible to the effect of this drug. In the East four times as much cocain and epinephrin is used as we believe to be necessary in the South. I never use over a 4 per cent. solution of cocain in my work. When I do a long operation, such as a submucous resection, I use this drug in combination with antipyrin; this gives complete anesthesia for three or four hours. Again, I never operate on a patient in whose case I expect to use a great deal of cocain without giving him a cup of black coffee before the operation. Coffee I think is one of the best antidotes against toxemia resulting from the use of cocain. I have learned this from doing operations under local anesthesia; this idea, I think, originated with Dr. E. W. Day of Pittsburgh.

I have stopped using gauze and the Berney's splint, and I never use the finger-cot with packing. I use the flexible tubular nasal splint devised by Dr. B. D. Kyle. This gives drainage and proper ventilation and there is no obstruction to nasal breathing. After this splint is introduced the parts are widened with forceps until the desired pressure is obtained. In our work, as in no other surgical work, proper ventilation and proper drainage are required. Nothing is more disagreeable than to have the nose stuffed up for twelve hours or more with packing; with this tubular splint there is proper ventilation and drainage from the start.

DR. MARGARET L. NOYES, Boston, Mass.: I should like to ask if the doctor has tried the method of sewing the septum after submucous resection. It seems to me a clean surgical procedure, and no packing is required.

DR. J. G. PARSONS, Sioux Falls, S. D.: To avoid the unfortunate results from sepsis in these septal operations, it has been my practice, after the flaps are in proper position, to insufflate an antiseptic powder which makes a satisfactory coating over the line of incision. It must be non-irritating.

DR. EMIL MAYER, New York: I am perhaps responsible for what has been stated in regard to the results of the Asch operation and in what measure this operation has met with success. I do wish to protest against any slur on the method. In its day it was the best operation. I still feel that in certain conditions of nasal deformity the Asch operation is the operation of choice, and it can be done with good results if it is done properly. Harm comes from its misuse and not from its proper use.

DR. C. W. WILKOWSKE, Chippewa Falls, Wis.: During the last eighteen months I have used the following method: First, I make one or two applications of a 10 per cent. cocain solution with cotton swab; secondly, I apply epinephrin solution, 1:1,000, in the same manner; thirdly, I pack both sides of the nose lightly with gauze saturated with a 6 per cent. solution of quinin and urea hydrochlorid, leaving the packing in place from ten to twenty minutes. This insures a painless operation, accompanied by very little bleeding and is followed by no toxic conditions and no sloughing of the flaps.

DR. GEORGE W. MACKENZIE, Philadelphia: Dr. Welty's contention in regard to operating on patients in the prone position is supported by a great many other operators.

As a safeguard against cocain toxemia I always instruct my patients to eat a full meal before the operation, since toxic substances produce a heavier degree of toxemia when the stomach is empty than when it is full. We all know the effects of whiskey or tobacco on an empty stomach, especially when taken before breakfast. I advise the use of coffee as an aid in the prevention of cocain toxemia. For those who are accustomed to it I have, at times, advised smoking during or shortly after the infiltration; I believe that the smoking tends to relieve somewhat the toxemia of cocain. For the

V. 6
1913

severe cases of toxemia following the use of cocain, amyl nitrite acts quite well. I believe that not more than one out of twenty cases of suspected cocain poisoning are really true cases of cocain poisoning, especially if one considers the small amount of cocain used in Schleich's method of infiltration (1.5 grains to 100 c.c. of water) and that of this solution but a small portion is used, and that a still smaller portion enters the circulation, for the greater portion of that injected is held between the mucous membrane and the cartilage or bone. I take exception to what Dr. Ballenger said regarding adhesions, because there are some real adhesions which result in cases of fracture. If one will reason out the pathology of the condition that results from fractures, he will find that a proliferative inflammation follows a new formation of tissue, and with it come the adhesions. On account of the small amount of cocain used, I do not often find it necessary to operate with the patient in the prone position, although I do operate on patients in this position when it is necessary. With the sitting position the operator sees quite well and can do the operation better no doubt than if the patient is in the recumbent position. If the patient manifests any symptoms of cocain toxemia, he should at once be placed in the prone position until the symptoms have passed. I do not like to cut off a dog's tail by inches, but I find that formerly, when we used this combination method, we have a greater degree of reaction and more frequent infections than we have now by the method advocated in my paper.

I believe that the tonsils and adenoids should be removed in advance of the septal operation; I think that infection is prone to occur after a septum operation from infected adenoids and tonsils. It is surprising what a large number of patients, adults included, have adenoids or, at least, some thickening of the posterior pharyngeal wall. Some patients whose adenoids are not particularly large develop an acute inflammation of the adenoids when the nose is packed. Infection which had been present, but latent, has been awakened and becomes active and from this source the infection spreads to an accidentally made wound of the septum.

ETIOLOGY, DIAGNOSIS, PROGNOSIS AND TREATMENT OF SPHENOPALATINE GANGLION NEURALGIA*

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In several papers on the sphenopalatine ganglion I have outlined what seemed to me the rôle[1] which it plays in nasal headaches and called attention to the topographic anatomy[2] of the district in which it lies, citing that the pterygomaxillary fossa is surrounded above, in front, behind and within by nasal structures and that in this respect it resembles an accesory nasal sinus; but that it is filled by the sphenopalatine ganglion, blood-vessels and connective tissue, instead of by air. I stated that inflammation in the surrounding nasal structures (accessory sinuses, sometimes the nasal membrane) produced by its extension to or the transmission of its toxins to the ganglion, a symptom-complex, partly neuralgic (painful); and partly motor, sensory and gustatory.[3] The neuralgic picture is pain in the root of nose and in and about the eye, in the upper jaw and teeth (sometimes lower jaw and teeth) extending backward under the zygoma to the ear, frequently making earache and pain in the mastoid; but severest often at a point 5 cm. back of the mastoid; extending thence to the occiput, neck, shoulder-blade, shoulder, breast and, when severe, to the arm, forearm, hand and fingers; with sometimes a sense of sore throat on that side.[4] Rarer additions to this picture are itching of the skin of the upper extremity, taste disturbances (parageusia), a sense of stiffness and muscle weakness in the upper extremity and fortification scotomata. Mild cases are described as a sense of tension in the face and stiffness or "rheumatism" in the shoulders. It may appear as constant pain with exacerbations, or it may stop and reappear cyclically as a migrain; or it may stop and reappear with stabbing sharpness as a tic.

The motor phenomena are changes in the appearance of the soft palate. Its arch is higher on the affected side, and the dimple which forms in the raphe just above the uvula in the act of gagging is deflected to the well side. The uvula is inclined obliquely to the well side. The sensory phenomena are slight blunting of the tactile sense of the soft palate, pharynx and tonsils, with like condition of the membrane of the nose of the affected side. The gustatory phenomena are confined usually to a slight blunting of the sense of taste on that side. Parageusia is rare. I have never seen all the manifestations in one case.

ETIOLOGY

Recently I have described[2] the environment of the sphenopalatine ganglion and stated that I believed that this nerve center became involved by extension of the inflammatory process from the postethmoidal-sphenoidal cells or from the membrane of the nose. Further observation strengthens my belief in this conclusion. Since then I have seen some postethmoidal-sphenoidal inflammations continue active for two years, with and without the usual symptoms of such inflammation, in patients who refused to be operated on, and then establish a typical sphenopalatine ganglion neuralgia which could be controlled by local application of cocain and was stopped by injection of phenol (carbolic acid[5]) alcohol into the ganglion, despite the fact that the surrounding causative inflammation remained. At that time I had seen practically only this type of case. This is by far the most frequent. Since then, however, I have seen twelve cases of systemic toxic origin. These arose in patients already well known to me in whom there was not a preceding local surrounding inflammation nor a general coryza. Cocain applied to the sphenopalatine foramen was always effective in stopping the pain. In one patient I have seen this phenomenon in very great severity on four different occasions. She has had violent neuralgias in other parts of her body. She has never had any nose disorder, but she is highly "neurotic." These attacks were clearly of some systemic toxic origin. It seems to me they correspond to the different intercostal attacks that she has had. However, cocain applied to the ganglion has always stopped the pain of this origin just as it has that of local origin. In fact, I think that these cases of whatever severity are more easily con-

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

1. Sluder, Greenfield: Rôle of the Sphenopalatine (Meckel's) Ganglion in Nasal Headache, New York Med. Jour., May 23, 1908, pp. 989-990.

2. Sluder, G.: The Anatomic and Clinical Relations of the Sphenopalatine (Meckel's) Ganglion to the Nose and Its Accessory Sinuses, New York Med. Jour., Aug. 14, 1909, pp. 293-298; Trans. Am. Laryng. Ass'n.

3. Sluder, G.: Further Clinical Observations on the Sphenopalatine Ganglion (Motor, Sensory, and Gustatory), New York Med. Jour., April 23, 1910, pp. 850-851.

4. Dr. M. A. Bliss is at present considering explanations for this unusual reference distribution.

5. Sluder, G.: A Phenol (Carbolic Acid) Injection Treatment for Sphenopalatine Ganglion Neuralgia, THE JOURNAL A. M. A., Dec. 30, 1911, p. 2137.

trollable than those of local inflammatory origin, as might be surmised (the inflamed tissues not being so readily cocainizable); and are more apt to yield to simple cocainization or some tentative means.

In March, 1913, I found the text of W. Krause[6] which, although published in 1905, came under my recognition only then. In it he gives a description of the macroscopic anatomy of the sphenopalatine ganglion which is, so far as I know, unique, and may explain some of the clinical phenomena—an explanation of which I have not heretofore ventured. He states that instead of the single ganglion as usually described, it may be two ganglia, one suspended on each of the sphenopalatine nerves. He also states that it often protrudes into or through the sphenopalatine foramen to lie submucous to the nasal fossa, which fact explains how the above-described syndrome may be established in some patients by a coryza of ordinary severity without involvement of the postethmoidal or sphenoidal sinuses; and may subside on recovery from the coryza.

ETIOLOGY OF RECURRENCE

After the cure of the neuralgia a recurrence may be established in several ways. I believe that it makes no difference what means were used to cure the particular case.

1. The pain may be rekindled by inflammation around the ganglion just as it was produced primarily. In persons cured by injection such a recurrence is often transitory. At the time of a coryza they may have pain, which disappears spontaneously with the coryza; or it may be quite as severe and persistent from recurrent empymata, as the original condition, and require injection the second time.

2. Cases that were of toxic origin may be excited again by the same etiologic factors.

3. In very severe cases that were with difficulty controllable, the pain may be aroused again by mental shock, to the full severity of the original condition. I have seen this in four severe cases, one of which had to be injected again on this account.

4. In a small percentage of patients who have remained well after injection for three or four weeks, there recurs a moderate degree of pain, which is made worse by application of cocain and is soothed by application of an irritant such as silver nitrate over the sphenopalatine foramen. I do not know an explanation of this phenomenon.

5. Frequently I have seen pain recur in mild degree as a result of fatigue and disappear after rest.

DIAGNOSIS

The diagnosis of sphenopalatine ganglion neuralgia has called into consideration phenomena that heretofore have been inexplicable, an understanding of which I think I now have.

The above-mentioned syndrome may be produced by lesions of the nerve trunks which supply the ganglion,[7] namely, the second division of the fifth and the vidian nerves. It is supplied with sensory fibers by second division of the fifth. The vidian nerve is composed of the great superficial and the great deep petrosals, the former (great superficial petrosal) coming from the geniculate ganglion bearing motor fibers from the seventh and taste fibers which have arisen in the anterior two-thirds of the tongue and are to reach the brain by the fifth nerve[8]; the latter (great deep petrosal) being a sympathetic nerve, branch of the carotid plexus. Both the second division of the fifth and the vidian trunks frequently lie in very close association with the sphenoid sinus. They may be separated from the cavity in the body of the sphenoid by only an eggshell's thickness of bone; and this may be fact as early as the third year of life for the second division of the fifth, and the seventh year for the vidian. Each year of life presents the same possibilities, from childhood to maturity. I learned this from a recent inspection of the matchless material, property of Dr. Warren B. Davis, Keen Research Fellow of the Jefferson Medical College, Philadelphia. This material consists of 145 sections of the Caucasian head from two months fetal life up to maturity (twenty-five years) uninterrupted, several specimens for each year (except the eleventh—only one) showing the changes of each year. Therefore, when inflammation exists in the cavity which occupies the body of the sphenoid (the sphenoidal sinus or a postethmoidal cell) it may readily involve the associated nerve-trunks either by its extension or by its toxin passing through the thin separating bony wall. Two years ago I found that cocain readily passed through this wall and paralyzed the branches of the fifth[7] nerve. A lesion involving the nerve trunks central to a point of union of these trunks can reproduce or simulate the syndrome arising from a lesion of such a point of union. There exist, however, at this point of union, ganglion-cells. It is not a mere coalition of fibers. What part, clinically, these multipolar cells may play is as yet unknown; but the clinical fact remains that certain sphenoidal inflammatory cases simulate completely the typical neuralgia arising in the sphenopalatine ganglion.

The differential diagnosis may be made by the following facts:

1. Cocainization of the sphenopalatine ganglion stops the pain of a lesion in the ganglion proper.

2. It does not in any degree stop the pain created by the more central lesion of the nerve-trunks secondary to sphenoidal inflammation.

3. On the other hand, intrasphenoidal application of pain-reducing remedies, such as cocain, will under these conditions stop the pain—that is, a local anesthetic applied central to the ganglion is effective. In addition to these points of difference there is often a congestion at the site of the sphenopalatine foramen when the sphenopalatine ganglion is the starting point for the neuralgia. This is more particularly true for the cases of inflammatory origin. Cases of toxic origin usually show no change in the nose.

In summing up the various features of sphenopalatine ganglion neurosis[9] I mentioned all the phenomena I had seen up to that time. Since then I have seen two cases in able-bodied men which were accompanied by a marked sense of muscular weakness of arm and shoulder which vanished with the relief of the neuralgia. Once I saw a case of cyclical reappearance of this neuralgia accompanied by intense itching of the entire upper extremity. Ten times I have seen it appear as a tic douloureux of the above described distribution.

PROGNOSIS

The prognosis of sphenopalatine ganglion neuralgia is beset by several perplexities on which I have specu-

6. Krause, W.: Anatomie des Menschen, p. 547, Leipsic, Verlag S. Hirzel.

7. Sluder, Greenfield: Anatomic and Clinical Relations of the Sphenoid Sinus to the Cavernous Sinus and the Third, Fourth, Fifth, Sixth and Vidian Nerves, Trans. Am. Laryng. Ass'n, 1912.

8. Stewart, Purves: Diagnosis of Nervous Diseases, 3rd Ed., p. 142-143, E. B. Treat & Co., N. Y.

9. Sluder, Greenfield: Syndrome of Sphenopalatine Ganglion Neurosis, Am. Jour. Med. Sc., December, 1910; Tr. Am. Laryng. Ass'n, 1910.

V. 6
1913

lated. Frequently in severe cases that had been relieved by injection of alcohol, the benefit proved so transitory as to arouse the inquiry: Was it worth while? In contrast with these, however, are others of high grade which were easily cured by simple applications of cocain, formaldehyd, or silver to the inflamed sphenopalatine foramen district. Possibly all such variations could be satisfactorily explained were post-mortem material as frequently available in these cases as is the diseased lung or liver or heart. Possibly an exact knowledge of the anatomy of the ganglion in man (which is not known) would be helpful also. I feel, however, that I have a better understanding of some of these points now, from further clinical experience and a knowledge of work on collateral lines by May of London, although animal experiment, or post-mortem observation of my own is still wanting. The best treatises on anatomy give but a few words of description to its histology. The gray matter of the ganglion does not involve all of the sphenopalatine branches of the maxillary nerve, but is placed at the back part at the point of juncture of the vidian nerve,[10] so that many if not all of the fibers of the sphenopalatine nerves proceeding to the nose and palate pass to their destination without being incorporated with the ganglion mass.

> The ganglion may also receive fibers from the glossopharyngeal nerve conveyed to it through the small and large superficial petrosal nerves; this connection is sometimes described as a third sensatory root.[10]

The fact that herpes has never been seen as a part of this syndrome would seem to bear out this point, for herpes is never produced by lesions of sensory nerve-trunks; but only by lesions of the sensory ganglion cells. The ganglion cells, therefore, would seem to be a part of its motor and sympathetic attributes, which are constituted by the vidian nerve. It seems true from clinical experience, however, that the vidian nerve carries fibers which transmit the sense of pain also. Certain noses have been propitious for cocainization of the tissues internal to the assumed position of the ganglion with the effect of stopping entirely the pain of posterior distribution; and then, when the applicator was placed external to the assumed position of the ganglion the pain of anterior distribution stopped. When placed in the center it would stop both anterior and posterior pains. In other words, it would seem that the trunks of the nerves supplying the ganglion in these cases could be cocainized separately. This has seemed true also for injection and electrical (faradic) stimulation. Alcohol injected on the vidian side of the ganglion has produced great pain of posterior distribution: injected on the side of the second division of the fifth it made the anterior pain. The needle in these positions was attached to the faradic current, the other pole in the hand, with the same results.

The recent work of Dr. Otto May[11] seems to me to explain some of the transitory results of injection. He proved experimentally that the cells of the gasserian ganglion in the goat, cat and dog are not at all readily destroyed by alcohol; that they are quite resistent to its action even though the ganglion were surgically exposed and the injection put directly into its substance. He also proved that the nerve fibers are much more vulnerable to alcohol than the cells; but that the injection must be made exactly into the nerve trunk. When the alcohol was put merely around the nerve its effect, although marked, was transitory—the nerve quickly recovered its function.

So it would seem that inasmuch as the ganglion is small (5 mm.) and difficult of access, that we may assume that the transitory results have followed the instillation of alcohol into its environment rather than into its substance; and that the highly satisfactory results have followed the exact instillation of the alcohol into its midst, or into the trunks supplying it; and that the results may not be permanent even under these circumstances because these tissues are so difficult of destruction. (Whatever made the pain in the original cell, one may suppose, may make it in the regenerated cell.) And, on the other hand, the severe cases from which the patients have recovered so satisfactorily from surface applications to the membrane covering the sphenopalatine foramen and its immediate surrounding (whereby an inflammation was allayed), we may assume to have been produced by irritants to the cells and fibers without their actual structural involvement, their power of resistance being great, as shown by Dr. May. In this I am assuming these cells and fibers to be alike with those observed by Dr. May. These assumptions may likewise explain why the complete clinical picture has never so far been observed in a single case. The complete picture would require the complete involvement of all the cells and fibers of the ganglion alike, which in accordance with the above assumptions would not be apt to happen.

TREATMENT

In the treatment of these cases various remedies and several surgical means have been employed. Applications were made to the region of the sphenopalatine foramen of 2 per cent. solution of silver nitrate, 0.4 per cent. solution gaseous formaldehyd, 0.5 per cent. phenol with 0.1 per cent. iodin as a wash. In the more severe and stubborn cases injections of phenol-alcohol were used and in the worst the patients were operated on (intranasally) with the intention of removing the ganglion. But treatment has heretofore had only passing mention.

ANATOMIC CONSIDERATIONS

The treatment of these neuralgias has always been beset by difficulties which seem to me occasioned by the anatomy of these parts. The simple painting of the region of the sphenopalatine foramen will often be found difficult because of an irregularity on the septum, or the configurations of the lower turbinate, or both. Certainly, in a nose which presents a straight septum, a wide caliber nasal fossa, and a straight lower turbinate there can be no difficulty in reaching accurately the region of the sphenopalatine foramen. In such a nose, a needle bent at a right angle 0.5 or 0.66 cm. from its end could be introduced along the septum, to a point 0.33 cm. posterior to and slightly above the posterior tip of the middle turbinate; and could then be turned to a point outward which would bring its point to the membrane covering the sphenopalatine foramen. This could readily be punctured, thereby bringing the needle's point directly into the pterygomaxillary fossa at the site of the sphenopalatine ganglion.

This would seemingly be the simplest and best method of injecting medicaments into the ganglion. But even were this always a possible technic, it would still fail at times because the sphenopalatine foramen is sometimes placed posterior to the anterior surface of the pterygoid process, opening as it were into the vidian canal rather than the pterygomaxillary fossa.

10. Quain: Elements of Anatomy, Vol. III, Part 2, p. 22, Ed. 11: Longmans, Green & Co., London, 1909.
11. May, Otto: Functional and Histologic Effects of Intraneural and Intraganglionic Injections of Alcohol, Brit. Med. Jour., Aug. 31, 1912, p. 465.

Such a pterygoid process is V-shaped, apex forward. Injection under these conditions would be into the vidian nerve and not into the ganglion. It is for these reasons that I should like to call attention to the method of injection by means of the straight needle, which may be done in practically all noses, however irregular or narrow, and, under control, is almost infallible in its accuracy. The control consists in measurement of how far back the posterior tip of the middle turbinate the ganglion lies, irrelevant to the pterygoid process. The pterygomaxillary fossa is constantly reached about at its center, 0.33 cm. back of the posterior tip of middle turbinate; and it is this fact that I utilize in the therapeutic injection. Should the surgeon rely on the buttress of a solid pterygoid process to stop his needle when it is being pushed backward, as might seem perfectly feasible under the rule of the anatomy of the pterygoid process, he will find himself disappointed frequently because the pterygoid process is hollowed out by a prolongation of the sphenoidal sinus downward into it as far as the bifurcation of the plates. The needle then crosses the pterygomaxillary fossa and penetrates a thin film of bone to enter the sphenoidal sinus to meet with firm resistance only in the posterior wall of the sinus, or possibly not even there. It might readily enter the cranial cavity. The injection will then be into the sphenoidal sinus or cranial cavity. The sphenoidal sinus is sometimes set so far down in the body of the sphenoid that its lower aspect lies below the line of the needle thrust. Under these settings the needle enters the main cavity of the sphenoid as soon as it crosses the pterygomaxillary fossa.

The relation of the pterygomaxillary fossa to the posterior tip of the middle turbinate is one of the most constant in the nose. Even though the anterior aspect of the pterygoid process be V-shaped and be relatively forward of the opening of the sphenopalatine foramen it disturbs this relation little or none. The posterior tip of the middle turbinate marks the anterior limit of the sphenopalatine foramen. The pterygomaxillary fossa must of course needs lie external to the plane of the sphenopalatine foramen. These relations being borne in mind, it must appear that a straight needle introduced into the nose from the nostril to pass under the posterior tip of the middle turbinate at its origin from the lateral wall, in a direction backward and upward and slightly outward, must pass out of the nasal fossa into the pterygomaxillary fossa and enter the sphenopalatine ganglion or its immediate vicinity. The distance from the point of entrance of the needle so placed and ganglion tissue is almost invariably 0.66 cm.

The lateral wall of the nose plays an important part in this technic and its variations should be borne in mind: [The author gives these in detail in his reprints and in the Section Transactions].

INSTRUMENTARIUM

For the injection of the sphenolpalatine ganglion I use a simple straight steel needle 1 mm. in diameter. This is usually of considerable strength; and fastened in a heavy crossbar enables the surgeon to secure it in a strong grasp and put great pressure on it. My associates, Drs. C. A. Gundelach, H. E. Miller, and W. E. Sauer,[12] have suggested (and employed) a needle with a trocar and flange or ring from 0.5 to 0.66 cm. from its point to prevent too deep placement or insertion.

12. Dr. E. M. Holmes of Boston described a similar needle in an article entitled "The Intranasal Treatment of Meckel's Ganglion," Trans. Am. Laryn. Rhinol. and Otol. Soc., May 9, 1913.

TECHNIC

From the preceding description it is evident that a straight needle introduced through the nostril in a direction backward, upward and slightly outward, approaching the lateral wall of the nose at a point in the middle meatus marked by the origin of the posterior tip of the bony middle turbinate, arrives almost at once on the anterior wall of the pterygomaxillary fossa. Should its point now be pushed backward 0.66 cm. it will usually enter the sphenopalatine ganglion. Experiment has proved, however, that it is better to enter the point of the needle 2 mm. anterior to the posterior tip of the middle turbinate. Pushed backward from this point it proceeds in a direction slightly more upward and more outward than it would from the point farther back, and thereby it strikes more nearly the center of the ganglion.

It may appear that the correct placing and insertion of the needle is a bit of technic of easy and certain execution. It has not, however, proved so in my hands. A number of specimens operated on show the possible failure. (Detailed description omitted for lack of space).

The lower turbinate frequently interferes with an easy and accurate placing of the needle point. This happens readily when the turbinate is placed somewhat higher rather than lower on the lateral wall. Under such conditions, the tip of the nose must be raised well up before a straight line may pass over the upper convexity of the body of the lower turbinate to reach the desired point under the posterior tip of the middle turbinate; and sometimes even then considerable downward pressure will have to be exerted on the lower turbinate by the needle as it passes backward in order to permit it to reach its destination.

Irregularities on the septum may interfere with the passing of the needle backward. They are apt, however, to be individual for that particular nose. The tubercle of the septum lies above the line sought by the needle.

I prefer the simple straight needle to be inserted under slight or great pressure, as the case may be. Under slight pressure it is very easy to measure 0.66 cm. distance insertion; but when it requires great pressure it is by no means easy to estimate this distance. For this reason I prefer to withdraw the needle when I think I have gone the right depth. While the needle is out I see that it has not been plugged with bone—that it is open—and then reinsert it into the opening it has been withdrawn from. On reinsertion the correct distance may be measured exactly because it requires no strength or pressure to replace it; 0.5 c.c. 5 per cent. phenol in 95 per cent. alcohol is then injected.

It is my practice to rest the hypothenar eminence of my hand on the patient's chin. In this way strong and controlled pressure may be put on the needle. It is desirable that it should not jump across the pterygomaxillary fossa, but proceed backward cautiously. Should the wall be hard and straight and not possible of penetration by pressure thus applied, the needle may be taken firmly by the cross-bar mentioned above and thereby rotated to and fro through a semicircle from side to side, using it as a hand drill to penetrate the bone. The lateral wall may possibly be too hard to be penetrated this way. Should it be, a machine drill or burr will have to be employed (see above). In the three attempts to remove the sphenopalatine ganglion I used this method of removing the lateral wall anterior to it.

I have already spoken of applications of silver nitrate solution and formaldehyd made over the membrane covering the sphenopalatine foramen (which is usually inflamed). Often this suffices to stop the pain. When it

V. 191

does not, I recommend that the ganglion be injected with a 5 per cent. phenol solution in water or in 95 per cent. alcohol. Formerly, I injected approximately two or three drops of this solution and very often it sufficed; but the number of failures seemed to me unnecessarily large. For this reason, I have gradually increased the amount of the injection to 0.5 c.c. This larger amount is more successful, possibly for the reason that the ganglion is small and probably frequently missed, to be influenced by the solution placed in its neighborhood. Alcohol alone, as advocated by Schlösser[13] in 1893, used for this purpose is intensely painful in these parts. For this reason I have added 5 per cent. phenol to it, which renders it painless in small injections—three or four drops. In 0.5 c.c. amounts it is sometimes followed by a slight sense of pain, which the patient usually recognizes as different from his neuralgia, and which lasts for from two hours to three days. I have used 5 per cent. phenol in water with possibly less satisfactory results than the alcohol combination. No untoward consequences have followed this procedure. This treatment has been highly satisfactory in the great majority of cases. Rarely, however, there recurs, in severe cases, a considerable degree of pain after from four to six weeks of comfort. In these cases, a rather strange phenomenon appears, namely, that cocain applied to the region of the ganglion is at once painful, and the deeper the cocainization the more so it becomes. And, on the other hand, irritants such as stronger solutions of silver nitrate are soothing. I have seen five cases of this type. This phase was transitory.

CLINICAL PROCEDURE

In practice I see what results are to be given by local applications. I always allay the pain with cocain which in new cases is often curative and follow this by application of formaldehyd or silver. When this fails to give permanent relief I inject the ganglion with 95 per cent. alcohol to which 5 per cent. phenol has been added. And I reinject it when the result proves insufficient or on recurrence of the pain, using cocain as my guide. Should the pain be aggravated by cocain I do not inject phenol-alcohol into it. In the one case in which I did inject such a ganglion, the alcohol, although immediately soothing, was later followed by great intensification of the pain.

SUMMARY

It would seem from the foregoing text that the clinical picture is usually rather clear, that some technical difficulties beset the management of the case, that the multipolar cells of the ganglion may from analogy be assumed to be resistant to chemicals, and that this assumption may explain the varying behavior of these cases; but that the treatment is scarcely less satisfactory than treatment of unmixed trigeminal neuralgia or tic. Removal of the sphenopalatine ganglion seems to me to offer less in prospect than removal of the semilunar ganglion (gasserian). It seems to me the issue here is far more complicated. With removal of the semilunar ganglion the pain usually (not always) stops. When the result is once right it would seem difficult to have that district again involved by local causes. It is too far central. But the sphenopalatine ganglion, were it completely removed, still leaves its supplying nerve-trunks exposed to detrimental association with the sphenoidal sinus; and inflammations of the sphenoidal sinus district by central involvement of its nerve trunks may reproduce the original picture.

The preceding text consists of observations drawn from experience with 214 cases including 311 injections, as well as experiment on the cadaver. Sixty injections were done as a nerve-blocking preliminary to Hajek's postethmoidal-sphenoidal operation. In one case under nitrous oxid I encountered sufficient bleeding to delay the procedure for a few minutes. This patient had a slight secondary bleeding seven days later. In another case occurring in the beginning of my experience I made the troubles worse by what I later realized was an injection of alcohol into the sphenoidal sinus instead of the pterygomaxillary fossa. In another of my early cases I unfortunately injected a ganglion which became more painful on application of cocain. The condition was made worse.

GLAUCOMA

In connection with sphenopalatine ganglion neuralgia I feel that glaucoma should be mentioned. (Omitted in THE JOURNAL for lack of space).

Since the preceding text was completed Dr. E. M. Holmes[17] of Boston has reported forty-six cases treated by the injection of the sphenopalatine ganglion. [For discussion of this paper see author's reprint or Transactions, Section on Laryngology, Otology and Rhinology, American Medical Association, 1913.]

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ABSTRACT OF DISCUSSION

DR. H. W. LOEB, St. Louis: Dr. Sluder succeeded in drawing your attention to and educating you in understanding the influence of the sphenopalatine ganglion and the various pains about the face resulting therefrom, as well as the methods which have been developed in their relief.

It seems to me that there should be a more careful selection of the cases. Dr. Haskins of New York emphasized the influence of malposed teeth which stomatologists are able to relieve. Many patients come to the aural surgeon with symptoms referable to the nose; the skiagram shows malposed teeth and all the symptoms referable to the nose are entirely cleared up on removal of these teeth. In these instances relief is premised by some other method of procedure. There are many cases with almost unbearable pain resulting from conditions in the nose; these are entirely cleared up on removal of the teeth after skiagram has been taken which shows that these teeth are malposed.

There is one point to which I would take exception, the diagnostic indication for cocainization of the sphenopalatine region. For instance, in the nerve fibers coming to the sphenopalatine ganglion with its sensory impressions, there might be a point of irritation to which the application of the cocain might afford relief; but this does not stop the pain that is more distal, and that may be relieved. This condition is therefore not at all diagnostic unless the cocainization is done through the end organs. Cocainization of the sphenopalatine ganglion region in cases of these neuralgias causes a cessation of the pain if certain fibers in the nerve are carried there. This may be a mere quibble, but it merits consideration.

DR. CORYDON G. DWIGHT, Madison, Wis.: I should like to ask if any member of our association has met with cases of glaucoma and, if so, how many examinations he would make in getting Dr. Sluder's gross number. Dr. Sluder has found glaucoma more frequently than I have in these cases of sphenopalatine ganglion neuralgias and it may be because of my fault in making a diagnosis.

13. Schlösser: Erfahrungen in der Neuralgiebehandlung mit Alkoholeinspritzungen, Verhandl. des Kongresses für Inn. Medizin, 1907; Wiesbaden, J. F. Bergmann.

17. Holmes, E. M.: The Intra-Nasal Treatment of Meckel's Ganglion, Trans. Am. Laryng., Rhinol. and Otol., Soc., 1913. For discussion of this paper see author's reprint or Trans. Soc. Oto-Laryng., A. M. A., 1913.

DR. GREENFIELD SLUDER, St. Louis: Dr. Loeb spoke of Dr. Haskin's work, which seems to me irrelevant to the subject in hand. Concerning the point that Dr. Loeb raises regarding the diagnosis, it seems to me that there is no reason to assume that the point of irritation is some place distant from that which shows the irritation. This point was once raised by Dr. Scholtz of St. Louis, and since that time I have made the experiment many times, namely, cocainized some other point in the nose which showed no irritation. The result was uniformly a failure. Cocainization of the ganglion proper was uniformly successful in stopping the pain. Concerning what Dr. Dwight asked regarding glaucoma and its association with the sphenopalatine ganglion neuralgia: I have been on the lookout for it since 1908 and have seen it in each of the twenty-six cases that I have had the chance to observe. This observation was made primarily by Dr. A. E. Ewing of St. Louis in 1908, but since that time both Dr. Ewing and I have been exceedingly cautious in our statements bearing on these points. In all of these cases the pain of the glaucoma, however, has been uniformly controllable from the ganglion.

CHRONIC OBSTRUCTIVE MIDDLE-EAR DEAFNESS

GEORGE E. SHAMBAUGH, M.D.
CHICAGO

A great deal of confusion persists among practicing otologists regarding the diagnosis and especially the prognosis of those diseases of the middle ear which produce deafness as the result of obstruction in the sound-conducting mechanism. There is, perhaps, no form of ear trouble regarding the diagnosis and prognosis of which the patient has more just grounds for complaint than these cases of obstructive middle-ear deafness. The difficulty in diagnosing these conditions is sometimes increased by the combination of two distinctly separate processes; such, for example, as the development in middle life of a bony fixation of the stapes in a case in which there has been in childhood a long-standing middle-ear inflammation.

As regards the prognosis of obstructive middle-ear deafness, confusion has often been increased because of the effort to apply in the classification of these cases terms which a more recent study of pathology have rendered more or less obsolete. For example, the term "chronic catarrhal otitis media" has heretofore been applied to a rather large group of ear cases. This group included all those cases of chronic deafness resulting from obstruction in the sound-conducting mechanism which are not the direct result of residue in the tympanum from a chronic suppurative otitis media. Chronic catarrhal otitis media included, therefore, those cases of middle-ear deafness which result from the occurrence of the non-purulent middle-ear inflammations, and in which there are, as a rule, periods of long-standing tubal occlusion. To this group the term "chronic diffuse middle-ear catarrh" was often applied. Chronic catarrhal otitis media included as well those cases of obstructive deafness in which the membrana tympani and the eustachian tube are found to be normal, that is the cases in which there exists no evidence of either tubal occlusion or of a previous middle-ear inflammation. In these cases which gave the typical tuning-fork reactions of obstructive deafness the obstructive process was evidently restricted to the region of the labyrinth windows. To these latter cases the term "chronic circumscribed middle-ear catarrh" was often applied. The difference between the chronic diffuse and the chronic circumscribed middle-ear catarrh was often further emphasized by applying the term "hypertrophic middle-ear catarrh" to the cases of chronic diffuse middle-ear inflammation and the term "primary sclerosing middle-ear catarrh" to the cases in which the obstructive process was restricted to the region of the oval window. It was in this way that the term "otosclerosis" came to be applied to cases of primary fixation of the stapes, because this fixation was supposed to be the result of a chronic, circumscribed, sclerosing middle-ear catarrh.

In our present-day classification of chronic middle-ear process we exclude, of course, all those cases formerly described as circumscribed, sclerosing middle-ear catarrh in which the deafness is the result of primary fixation of the stapes. We still retain the term "otosclerosis" for these cases, but we understand by otosclerosis not a form of sclerosing middle-ear catarrh at all, but a disease beginning in the bony capsule of the labyrinth and in the large percentage of cases developing primarily in the region of the oval window and resulting early in a rigidity and finally in complete bony fixation of the foot-plate of the stapes.

In a present-day classification of middle-ear process there would be a distinct advantage if the term "chronic catarrhal otitis media" were dropped, especially because of the confusion in the minds of many which this term is certain to perpetuate. In its place might be used some such term as "chronic simple otitis media" or "chronic non-purulent otitis media." This term would convey at once an impression of the fundamental pathology of these cases, which is that of infection in the membrane lining the middle-ear chamber, with round-cell infiltration and thickening and the subsequent formation of fibrous connective tissue. This process is prone to acute exacerbation from time to time, usually associated with an acute infection in the nasopharynx. At these times secretion can, as a rule, be detected in the tympanum, not by the inspection of the drum membrane, because this structure is usually rendered too opaque from the chronic thickening, but by the inflation of the tube. This chronic middle-ear process may or may not be associated with a persisting tubal occlusion. In the cases in which the tubal occlusion has been a conspicuous factor there is sure to be a more or less marked retraction of the drum membrane. In other cases in which the occlusion of the eustachian tube has never been a conspicuous factor there is but little evidence of a retracted drum membrane. It is in these latter cases that the diagnosis from primary fixation of the stapes, otosclerosis, may not at times be so readily made. The membrana tympani, even in these cases, when the drum membrane is not retracted, usually appears more or less thickened and opaque; enough so, at least, to indicate the type of process involving the membrane lining the tympanum. The defect in the hearing in these cases arises from the obstruction in the conducting mechanism in the tympanum. The most pronounced disturbance in hearing arises because of the fixation of the stapes. When the tubal occlusion has been a conspicuous factor, the defect in hearing is often markedly increased by each acute exacerbation, which brings on a fresh closing of the tube. In such cases there is usually a marked improvement in hearing from a single inflation. On the other hand, the degree of retraction of the drum membrane is no index of the defect in hearing or of the improvement to be expected from inflation. It often happens that the occlusion of the tube which brought on the retraction of the membrane has long since disappeared and the drum membrane has been left

V. 191

in a retracted position. In these cases it is not unusual to discover no defect in the hearing.

Why it is that certain patients subject to recurring middle-ear infection, even with marked tubal occlusion, will suffer but slight permanent defect in the hearing while other patients subject to the same middle-ear infection, even though the tubal occlusion has been absent, develop serious interference in the conduction of sound-waves through the tympanum, is an interesting problem. The difference seems to hinge on the development of inflammatory adhesive bands in the tympanum. These bands are apparently the chief cause of the deafness. They connect the ossicles and the membrana tympani to the surrounding walls and are the result of the thickening during the acute inflammation of the membrane lining the middle ear. It seems probable that these thickened adhesive bands in the tympanum are often formed out of the folds of mucous membrane connecting the ossicles with the walls of the tympanum. Such folds vary a great deal in different individuals, being much more developed in some than in others. It is quite possible that this difference in the development of folds of mucous membrane in the tympanum is the fundamental reason why one person with recurring attacks of mild middle-ear inflammation develops a pronounced obstruction in the conducting mechanism, while another with a similar process suffers but a slight defect. A study of the normal folds of mucous membrane in the tympanic cavity takes on, for this reason, an added interest. The folds form, with the fan-shaped ligament placed around the neck of the hammer, very often a more or less complete partition between the attic and the cavum tympani proper. Accessory folds are most frequently found connecting the crurae of the stapes with the niche of the oval window. An additional fold is often found connecting the long process of the incus throughout its entire length with the posterior wall of the tympanum. Normally these folds show simply the two layers of flat epithelial cells with a few capillary blood-vessels lying between them. The folds are normally quite flaccid and do not interfere with the movement of the ossicles. When the membrane in the tympanum is subject to repeated attacks of inflammation these folds undergo the same process of thickening and stiffening as does the membrane lining the tympanum and in the cases in which the folds are well developed they must constitute an important factor in producing a rigidity of the conducting mechanism.

PROGNOSIS

The prognosis as regards progress of the deafness is on the whole better if the occlusion of the tube has disappeared and the existing defect in the hearing is dependent on changes in the tympanum which has ceased to be active, as the now patulous tube often indicates. On the other hand a persisting tubal occlusion usually indicates a process which is still active and one in which there will very probably be a further increase in the deafness. Another factor in the prognosis which must not be lost sight of is the development, in long-standing cases of middle-ear obstruction, of secondary degenerative changes in the cochlea. The presence of secondary changes in the labyrinth always makes the prognosis worse. The presence of these secondary degenerations in the internal ear can seldom be detected by either the Schwabach or the Rinné test, since in these cases both of these tests will continue to give the typical reactions of obstructive deafness. The labyrinth defect can best be detected by noting the defect for the higher notes of the Galton whistle.

Generally speaking, the chances of improving the hearing are less in those cases in which the occlusion of the tube has disappeared, or in which the tubal occlusion has never been an important factor, and the defect in hearing is dependent on the existence of adhesive bands in the tympanum. On the other hand, the defect in hearing which is found in cases of persisting occlusion of the tube, especially if secretion in the tympanum is present, is, as a rule, more readily improved by treatment.

CAUTION TO BE OBSERVED

It is highly important in the treatment of these cases to correct those conditions in the nose and throat which tend to keep up the middle-ear process, especially the presence of adenoids in the nasopharynx. The recognition of the conditions in the nose or throat which may be a factor in causing the middle-ear trouble, requires a careful examination and the exercise of considerable judgment. The indiscriminate operating on every anatomic variation which may be detected in the nose and throat in the hope of improving the hearing in these cases, should be discouraged. It is unusual for the middle-ear process to be kept up by conditions in the nose that can be improved by intranasal operations. On the other hand, the presence of adenoids in the nasopharynx always requires operative treatment, as do the faucial tonsils when these structures are subject to repeated attacks of acute infection.

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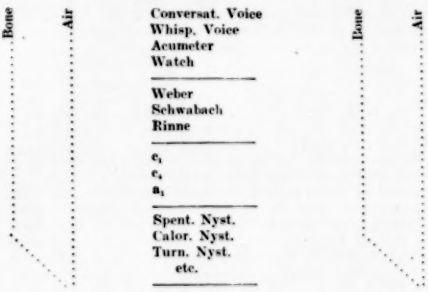
ABSTRACT OF DISCUSSION

DR. J. HOLINGER, Chicago: As a whole, this subject is usually neglected. The examination of the semicircular canals in suppurations of the middle ear, which is taught in Vienna and other places, is given such an enormous amount of time that other conditions within the ear receive but scant consideration. Cases of hard hearing, nevertheless, are so much more frequent than the suppuration of the labyrinth that I think the ratio of 1 to 500 is not overdrawn. Many cases of different forms of hard hearing are overlooked or are not discovered because the proper technic is not employed in examination. This technic is not easier or more simple than that of the examination of the semicircular canals. We sometimes fail to realize that the physician with the widest experience in the pathology of hard hearing has examined during life, as well as by means of the microscope after death, probably not more than 130 or 140 ears. This is quite a large series. There are people who have not examined more than twenty-five or thirty ears, mostly post mortem, and their opinions are compared with those of the more experienced men. These facts should be taken into consideration in weighing evidence in favor of or against certain teachings of technic of examination, especially tuning-fork tests. I should like to call attention to one thing that occurred to me in these cases of obstructive deafness, as Dr. Shambaugh calls them: the hypertrophy of the posterior end of the lower turbinals. The normal turbinal reaches to only within several millimeters of the normal opening of the eustachian tube. Hypertrophy of the posterior end often covers the opening like a valve. Posterior hypertrophies of the lower turbinals most frequently cause hard hearing.

DR. CULLEN F. WELTY, San Francisco: I wish to take exception to the nomenclature that Dr. Shambaugh has used, as it is confusing and misleading. Nomenclature that comes from definite pathologic lesions should be used, with subdivision when necessary. There are three great forms of deafness: (1) adhesive processes, (2) otitic sclerosis, (3) labyrinth deafness. All the mechanical hindrances naturally fall under the subdivisions of adhesive processes. Nomenclature

should be adopted, as we would then know what others are talking about. Sometimes two forms of deafness are combined in the same individual. At times it is more or less difficult to separate an adhesive process from a beginning otitic sclerosis, but after repeated examinations with the forks I am sure that one will be able to differentiate beyond a question of doubt. The patients who receive the most benefit do so through operative interference. In other words, the patients cure themselves when the trouble has been removed; especially so with "bubbles in the ear."

DR. GEORGE W. MACKENZIE, Philadelphia: Dr. Shambaugh referred to the lack of assistance afforded by the tuning-fork tests in the differentiation of pure obstructive deafness from those cases of obstructive deafness with secondary internal ear deafness. A further suggestion came from Dr. Holinger of Chicago, when he referred to our lack of thoroughness in making functional tests of the hearing. The inner ear may suffer, more or less, in pronounced and long-lasting cases of deafness from obstructive diseases, as pointed out, long ago, by Alexander and others. Microscopic sections show degenerative changes in the cochlea with partial destruction of Corti's end organ. The pathologic changes in the inner ear can be determined clinically, more or less, through the careful application of our tuning-fork tests, especially the Rinné test. Those who are familiar with the method of recording the functional test findings in the old Politzer Clinic will recall the following features: On the left of the schema was represented the findings of the right ear, and on the right the findings of the left ear. In other words, we looked at the records as though we were looking at a patient facing us. In the middle of the schema was printed:



Under the right and left ear and to the sides were dotted lines runing vertically, one shorter than the other, to represent graphically the normal Rinné test. The longer dotted line represented the longer hearing from air conduction, and the shorter dotted line the shorter hearing over the bone. In the case of normal bone and air conduction, these dotted lines could be filled in, representing the normal findings, that is, positive Rinné, with bone conduction as long, but no longer than the normal (used as a control), and air conduction as long as the normal. In the case of a pure obstructive lesion we find the air conduction shortened and the bone conduction lengthened, and this lengthening is in proportion to the shortening of air conduction. This is quite mathematically so, as can be shown. In cases of marked obstructive disease we find the bone conduction so lengthened that it is actually longer than the normal. This may appear paradoxical; nevertheless it is so, and can be proved by experiment. In the event of such a patient developing secondary internal ear affection, the bone conduction begins to shorten. It need not become shorter than the absolute normal, but a relative shortening is a proof of secondary internal ear involvement. In some cases the shortening may be so marked as to bring it to the normal or even shorter than normal; then the diagnosis becomes quite easy. If a patient, for instance, shows a negative Rinné with a bone conduction equal to that found in a normal individual—knowing that bone conduction must be lengthened in a pure obstructive disease—it requires no stretch of the imagination to determine that we have here a case of obstructive disease with complicating inner ear affection.

DR. GEORGE L. RICHARDS, Fall River, Mass.: I recently saw a patient about 36 years of age who complained of a stopping-up in the ears. He was relieved after treatment, but only for a short time, and returned every three or four days. I never thought of the possibiliiy of adenoids, at first, as there was no difficulty in breathing and the catheter was easily passed. As there was no permanent improvement, I examined him postnasally and found a large amount of adenoid tissue. This was removed and his hearing was at once permanently improved. We are often put off our guard because of the age of our patients and do not make the examination with the care we should. Diagnosis is difficult even when great care is used and tuning-forks as well. A man who has studied these cases and made many post-mortem examinations of the cases that he had examined clinically during life, said he was unable, even with the tuning-fork tests, to make an accurate diagnosis.

DR. J. C. BECK, Chicago: So far as my experience is concerned I think that in cases of open tubes practically nothing is done to improve other conditions, whether in the nose or elsewhere. I have found, in cases in which the tube was not entirely blocked, but an irritated condition was being perpetuated in that region not ordinarily seen by the mirror, that if the examination is made after retracting the soft palate, masses of adenoids in the posterior pillar can often be seen. With the mirror as ordinarily used the parts do not come into sufficient view. These adenoids do not necessarily obstruct, but they are irritating and influence this condition. This is the view held by Dr. Shambaugh and Dr. Holinger.

DR. CORYDON G. DWIGHT, Madison, Wis.: In closing, I wish Dr. Shambaugh would give us something besides the operative treatment.

DR. GEORGE E. SHAMBAUGH, Chicago: The chief point I wish to bring out is that we must make more of a difference between these cases of obstructive middle-ear deafness than is often done. Too often it has been the practice to treat all cases alike; that is, to keep on treating patients whether there is any chance of improving the hearing or not. A careful study of these cases will usually make it possible to differentiate between those that are likely to be improved by treatment and the ones in which treatment is of no avail. In doubtful cases always give treatment a fair trial, but a fair trial does not mean treatment for a month or two. If the case is one that will be improved by treatment, improvement can be detected after a few inflations—about a half dozen treatments. In the prognosis, it is of considerable value to determine the evidence of a secondary degeneration in the labyrinth. This cannot be determined by either the Rinné or the Schwabach tests, but such a degeneration can be detected by examining the upper tone limit. There has been a great deal of unnecessary surgery performed in the nose in the treatment of deafness. It requires judgment to determine the conditions in the nose that may influence unfavorably the progress of a middle-ear process. In general, I should say that entirely too much importance has been attached to the effect of anatomical variations in the nose on cases of obstructive middle-ear deafness.

So-Called "Recurrence" of Cancer.—Now comes the question, How far are you going to trust your sense of touch for the diagnosis of carcinomatous lymph-nodes? Remember if you make a mistake on this point and remove a carcinoma on the side of the tongue, which is a small matter, you have left that patient with carcinoma, and do not try to soothe your mind by saying, when the lymph-nodes continue to grow, that she has some recurrent disease of the lymph-nodes in the neck. I do not know exactly what this word "recurrence" means, but I think it is simply a cloak for carelessness and ignorance.—C. B. Lockwood in *Clin. Jour.* (London).

TEMPOROSPHENOIDAL ABSCESS WITH MENINGEAL SYMPTOMS

DRAINAGE THROUGH THE CISTERNA MAGNA; MASTOID OPERATION; RECOVERY

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General purulent meningitis presents many problems of interest to the otologist. This condition is still frequently overlooked when it occurs as a sequel to purulent foci in the middle ear, as shown by the statistics of Kittredge. The diagnosis of general meningitis is at times attended by the greatest difficulties, even when the patient has the advantage of hospital care and can command the services of the various experts connected with other departments of medicine.

In dealing with this condition as a surgical problem the most radical conservatism and experience in this particular procedure as well are prerequisites for a successful issue.

The case that I am about to report may or may not have been one of general meningitis. Several points in the history as well as the physical examination of the case pointed strongly to a general meningitis. The surgical procedure by means of the Haynes operation showed that drainage through the cisterna magna is not attended by great difficulties or danger to the patient. A large number of cases, however, will be necessary to settle the question of mortality.

History.—A. McF., aged 22, single, day laborer, born in Scotland, was admitted to the outpatient department of the Massachusetts Charitable Eye and Ear Infirmary in the service of Dr. Plummer Oct. 26, 1912. He was sent into the house immediately and the following history obtained from his sister:

A discharge from the right ear was continuous until he was 14 years old. It was then quiescent with only an occasional temporary discharge, until one year ago. Since then the discharge has been more frequent. This last week there has been a constant purulent discharge with pain, headache, vertigo and vomiting. Sore back and neck with cramps in the stomach one day. Left ear occasional discharge.

Examination.—Patient well nourished and cheeks flushed. He repeats constantly, "Oh, my poor head," so that no direct answers can be obtained to questions. Sensorium cloudy. Patient has marked rotary nystagmus to the right. Pupils equal and react to light. Marked rigidity of the neck. Knee-jerks absent. Marked Kernig on both sides, also marked tache cérébrale. No Babinski or ankle-clonus. Lumbar puncture done at once and 3 c.c. of cloudy fluid withdrawn. Pressure not marked. White count 31,400. Blood-pressure 125. Urine negative. Cultures were contaminated and valueless. Eyes negative.

First Operation.—Ether anesthesia. On account of the marked turbidity of the cerebrospinal fluid, the rigidity of the neck, positive Kernig and tache cérébrale, cloudy sensorium and rotary nystagmus without focal symptoms, it was decided to do the Haynes operation for decompression and drainage through the cisterna magna. Accordingly the usual operating-table, with the head extension lowered, was utilized. The patient was etherized in the usual way, then turned over on his chest with the head well flexed and supported on each side, and the etherizer under the table.

An incision was made in the median line from the occipital protuberance to the spinous process of the axis and carried down to the occipital bone. The periosteum was reflected and the skull trephined an inch above the margin of the foramen magnum. A button of bone was removed. From this opening extending downward and outward on each side a triangular area of bone was removed about three-quarters of an inch wide at the base near the foramen magnum. After this wedge-shaped piece of bone was removed the dura was exposed and found bulging moderately. The occipital sinuses were not recognized. The dura was incised in the median line for about 2 cm., followed by a slight escape of spinal fluid. After the dura and arachnoid were opened the cerebellum prolapsed into the incision to a slight extent. At this stage of the operation the patient became very cyanotic and stopped breathing. He was at once turned over on his back, artificial respiration performed for several minutes and oxygen administered, after which he rapidly recovered. A cigarette drain was inserted between the edges of the incised dura and arachnoid into the cisterna magna for a quarter of an inch. The soft parts were allowed to fall together and the muscles were united by four silkworm-gut sutures. A large sterile dressing was applied with a retaining bandage. A shock enema and strychnin sulphate 1/10 grain were given subcutaneously.

October 27.—Patient made a good recovery and is in fair condition this forenoon and it was decided to explore the right mastoid.

Second Operation.—Ether again administered. The usual mastoid incision was made on the right side. The periosteum was reflected and the cortex exposed and found to be very thick and non-cellular. After the cortex had been removed the pus welled up into the wound, but not under pressure. The antrum was found and cleaned out. After entering the middle ear the tegmen was examined with a probe. An opening was found in the roof, through which the probe passed, followed by a sudden flow of foul, semipurulent discharge, which welled up from the cavity and about two tablespoonfuls escaped. Good drainage was established through the tegmen by enlarging the opening and a large rubber tube inserted without washing or manipulation. This was held in place by one silkworm-gut suture. A sterile dressing and bandage was applied. Hexamethylenamin (urotropin) 7½ grains was given four times a day.

Postoperative History.—October 28: Patient seems more comfortable but complains of severe headache and backache. Both wounds dressed. There is considerable serous discharge from the tube in the abscess cavity.

October 29: The wick in dura was changed and shortened. The tube in the abscess cavity was shortened. Directions that the external dressings be changed three times daily and that the tube be kept patent without removal, that is twice after the morning dressing.

October 31: The patient is comfortable but restless. Large doses of hexamethylenamin continued. Each dressing shows a slight backing up of pus.

November 2: Hematuria is present and the hexamethylenamin was stopped. The patient is restless but not unconscious. He continues to complain of pain in the back of the neck and head. Projectile vomiting present in the evening.

November 3: Restlessness continues. Temperature 103. Pulse and respiration normal. During my absence a confrère tried irrigating the cavity with normal salt solution. Pupils almost normal and light reaction improved. No tache cérébrale seen.

November 8: Very little pus escaped from the abscess cavity on dressing. Patient has been comfortable for the last twenty-four hours. Takes nourishment well. Neck wound entirely healed (thirteen days after operation).

November 11: The discharge is more serous, being thin, watery and clearer. Patient has nausea occasionally. Pupils react well.

November 14: Patient much brighter. Eats heartily. No discharge from the abscess.

November 16: Patient continues to vomit at intervals. A few drops of pus oozed out of the wound yesterday at the dressing. Middle ear not dry.

November 18: Patient very comfortable except for pain in the neck. No vomiting for two days. Eats semisolids. Slight discharge from the abscess cavity together with small pieces of disintegrated brain tissue. External dressing changed twice daily. Patient very sensitive to any obstruction of drainage.

November 19: Patient allowed a bed-rest. After sitting up for ten minutes he became dizzy and vomited a good deal. This evening he is comfortable and has a normal temperature.

November 21: No further vomiting. Mastoid wound closing. Slight discharge from the middle ear.

November 26: Patient vomited a little yesterday, after eating a fairly hearty meal, the first time for several days. Feels comfortable.

December 3: The wick was left out of the mastoid wound yesterday. Slight discharge from the mastoid and middle ear. Patient sat up a few minutes with no bad effect. Slight vomiting in the afternoon.

December 7: Patient has been feeling well with no symptoms until today when he had hiccups and vomited after eating. No headaches.

December 10: Patient vomited yesterday and had hiccups but is comfortable to-day. Slight discharge from the middle ear. Mastoid wound nearly closed.

December 12: Patient much better. Sits up daily with no ill effects.

December 15: Mastoid well epidermatized. Dressing omitted. Wound of Haynes operation shows a few exuberant granulations which were touched with silver nitrate. Patient sits up forenoon and afternoon and is gaining in strength. Appetite good.

December 18: For two days the patient has vomited several times. No other disturbance. Middle ear has been dry for three days.

December 24: Patient walks up and down the ward. Feels well.

Jan. 2, 1913: Discharged to outpatient department. Hearing test shows:

R.		L.
1		18
—	W.	—
25		25
12"		12"
—	AC.	—
32"		R. 20"
	BC	
	+ LW.	

N.-Galton-N.
96 Low 96

Atypical Symptoms.—The patient was brought in at the close of the service. His symptoms were urgent. It would have been instructive to follow out the chemical examination of the cerebrospinal fluid as suggested by Kopetsky. Unfortunately even the infective organism was not determined owing to contamination of the specimen. The condition of the patient did not justify delay for further study. Dr. Haynes' indications for operative interference were not all present. The blood-pressure was not above the normal limits. There was no edema of the papillae. The absence of the carbohydrates from the cerebrospinal fluid was not determined. There was a marked clouding of the sensorium and in addition a turbid cerebrospinal fluid, marked rigidity of the neck, positive Kernig, marked tache cérébrale, rotary nystagmus to the right and general headache. If general meningitis was present it was undoubtedly in the early stages, where an immediate drainage of the ventricles promised the most hope to the patient. While a localized meningitis secondary to a brain abscess might cause a great many symptoms of general meningeal irritation, it was a fair inference that in this case a general meningitis might be present, although later developments made it doubtful.

Haynes Operation.—The mechanical difficulties of the operation are not many. Some annoyance from the persistent bleeding was experienced on removing the button of bone.

The margin of bone around the foramen magnum is thick and in the adult deeply placed under the skull. Neither of these difficulties is of enough importance to require extended comment.

IMPORTANT POINTS

Early Decompression.—The importance of early decompression need not be emphasized. The effect of increased intracranial pressure in depriving the vital centers of their blood-supply as well as the harmful retention of bacterial ptomains and toxins is well known.

Hexamethylenamin.—The value of hexamethylenamin, on which Cushing lays great stress, is dependent on sufficient circulation to enable it to reach the vital centers. Another requisite is an acid reaction of the secretions which is only present in the early stage of meningitis.

Difficulties in Drainage in Brain Abscess Cases.—No part of the after-care of a case of brain abscess requires more constant watchfulness than the drainage. Theoretically a cigarette drain after a careful and complete evacuation of an abscess cavity should be the ideal method. This permits the escape of the thin secretions and the leukocytes should do the rest. This is undoubtedly true when we have provided a free approach, so that the cavity can be completely evacuated, but when this is not done, or is not feasible, a large drainage-tube is better. When a tube is employed its lumen must be kept free, even if it is necessary to examine it several times in the twenty-four hours. The slightest blocking is apt to be followed by vomiting and increase in temperature.

Frequent manipulation of the cavity itself is to be deplored, but constant watchfulness of the drainage, which has been chosen to meet the requirements of the special case, is necessary and important.

No definite conclusions can of course be drawn from a single observation. The following points, however, I should like to emphasize as brought out from the study of this case:

CONCLUSIONS

1. The Haynes operation for drainage through the cisterna magna is not a difficult surgical procedure.

2. Further study is necessary to determine whether the Haynes or any other procedure is effective, except in the early stages of purulent meningitis.

3. This may depend on whether the infection is always accompanied by edema and serous effusion. It seems possible that, as a primary manifestation, there might be a plastic exudate, fibrinous in character, which would not drain until a later stage, when it became purulent. Even if this were so, a decompression operation might so relieve the intracranial pressure that the patient would have a chance to survive until the leukocytes could take care of the exudate.

4. The pressure of a large brain abscess makes the diagnosis of general meningitis in this case uncertain.

5. This case is reported in some detail to show also how a little blocking of drainage is followed by hiccups or vomiting, even as late as the third or fourth week, in cases of brain abscess.

6. Whether the drainage through the cisterna magna contributed to the successful outcome, may be an open question, it apparently did not add any danger to the patient.

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ABSTRACT OF DISCUSSION

DR. J. C. BECK, Chicago: I have had some experience in this line and only recently reported seven cases with six post mortems and I wish to repeat certain testimony in reference to these cases. My results show one patient living. Beyond any doubt, this was a true suppurative case. The cerebrospinal fluid showed the presence of the pneumococcus, and this type of meningitis ends in recoveries. There were two other recoveries from this form of infection. They may recover

V. 191

from such an operation or with spinal puncture alone. I have had one tuberculous patient, as Dr. Emerson had; my patient is dead while his is living. I made my diagnosis by means of every point laid down, as well as the chemical analysis by Dr. Kopetski. In this case, when the patient was turned over he stopped breathing. Twice during the operation I attempted to turn him over, with the same result. There was no doubt that something pressed on the vital respiratory center. I made no diagnosis of abscess at that time. I operated and made a mistake by taking too long in performing the operation. I did a simple but extensive mastoid operation at the same time. The patient died the next day. The post-mortem showed a temperosphenoidal abscess without any demonstrable evidences of perforation, but on close examination a chronic point of infection was found in the subdural tract. The interesting point about this case is that secondary diffuse meningeal abscesses may have acute exacerbations. This patient got on a "drunk" and the meningitis followed. The other five cases that I have had bring up the point that operation in the later stages of meningitis is inadequate. An exudate is present, and pockets for the retention of material, both at the base and about the convexity, which cannot be drained from an opening. The operation itself is not a difficult one. I believe, however, that this operation is going to be overdone. Many of the patients who are operated on recover, but these are cases of serous meningitis. Men who are not experts in this line of work may cause death from this operation when recovery would result in the hands of one properly qualified.

DR. GEORGE E. SHAMBAUGH, Chicago: I wish to emphasize a point brought out by Dr. Beck: Be sure of the diagnosis. It seems to me quite possible that in the case reported by Dr. Emerson the patient might not have had meningitis. An otitis media in a child will sometimes produce marked symptoms of meningitis which will clear up in a few hours after the rupture of the drum membrane. An examination of the cerebrospinal fluid does not always give the information we desire in doubtful cases. I have recently seen a case in a child in which a marked increase in the cellular element, together with a culture showing staphylococci in the cerebrospinal fluid was present, and post-mortem demonstrated a cerebellar abscess, but no meningitis.

DR. GEORGE W. MACKENZIE, Philadelphia: I am inclined to think that the cases that have been reported may not have been diffuse purulent meningitis. I believe Dr. Emerson himself did not absolutely claim them to be. In cases of abscess of the brain, for instance, we may get any and all kinds of symptoms. There are several stages: the initial stage, or that of invasion; the secondary stage of incubation; the third stage, in which the symptoms are quite manifest, and, finally, the terminal stage which precedes death. We have symptoms due to the toxemia and we may find a variety of symptoms suggesting some febrile condition from the pressure. We may find symptoms referable to distant parts. This was pointed out by MacEwen in 1893. An abscess on one side of the brain may cause pressure of the medulla against the opposite margin of the foramen magnum, involving both motor and sensory tracts. We may have almost anything in the way of symptoms in brain abscess. The cerebrospinal fluid in these cases is not constantly the same. When the abscess is encapsulated, we may find the spinal fluid but slightly clouded and free from micro-organisms. In other cases in which the abscess is superficial or has ruptured into the ventricles the fluid would be cloudy and would contain bacteria. There is a reasonable doubt in most cases of cerebral abscess and diffuse purulent meningitis, and we must depend solely on clinical findings.

DR. J. HOLINGER, Chicago: In *Ztschr. f. Ohrenh.*, I have published the history of a patient who recovered from typical meningitis. He died eight months later and the changes found at the post-mortem confirmed the diagnosis. Last spring, a girl, aged 18, was observed suffering from meningitis of unknown character. She had been operated on for tuberculous trouble of the external and middle ear about seven years previously. She complained of pain in her forehead, which disappeared after a few treatments. Two weeks later she became unconscious for three days. She improved, but suffered a relapse three weeks later, and was again unconscious for four days. She was afflicted with stiffness of the neck and would often cry out with the pain. At the lumbar puncture a clear fluid was discharged under pressure, which contained no microbes. When I operated I removed a button the size of a half dollar from the temporal bone, expecting to find a tuberculous abscess, but there was only a discharge under considerable pressure, of a large amount of clear cerebrospinal fluid. The discharge lasted for several days and the patient finally made a complete recovery. The diagnosis, from the evidence at hand, as well as by exclusion, was that the disease was probably of a tuberculous nature. Some forms of meningitis, at least, are amenable to operative treatment.

DR. WILLIAM L. BALLENGER, Chicago: I agree with Dr. Emerson that these were not true cases of diffuse purulent meningitis. I shall like to emphasize the possibility and probability of an involvement of the labyrinth. There is nystagmus to the right, possibly a circumscribed meningitis, with a high degree of hearing. There also is the possibility that there was no circumscribed labyrinthine disease, but a central toxemia caused by the pressure from an abscess. These things are interesting in a speculative way. I should like to ask how long the rigidity of the neck lasted. Was it immediately relieved, or did it last some days after the operation?

DR. BURT R. SHURLY, Detroit: The scientific use of hexamethylenamin has not been mentioned in connection with these cases. If you are to perform a lumbar puncture, or puncture the ventricle, while the cerebrospinal fluid is dripping inject into the bowel 10 or 15 grains of hexamethylenamin and within one or two minutes the chemical test will prove the presence of the drug in the cerebrospinal fluid. It is surprising and marvelous how quickly this absorption takes place from the bowel into the lymphatics.

DR. F. P. EMERSON, Boston: I agree with those who discussed my paper that this was probably not a case of general meningitis. The eyes were examined and found to be negative. The rigidity of the neck lasted about twenty-four to forty-eight hours and then at once cleared up. My object in reporting this case was to call forth some discussion based on those cases which have been operated on by the Haynes method during the last year, thereby hoping to come to some definite conclusion, as to whether a patient, after some time, can be relived by any operation. The post-mortem findings in some of Day's cases showed a thick exudate in the floor of the fourth ventricle and pockets of pus in remote districts. This makes us skeptical as to whether late drainage by any method is effective.

THE HEARING FOLLOWING THE RADICAL EAR OPERATION

MY IMPROVED TECHNIC: THIERSCH GRAFT VS. TAMPON

CULLEN F. WELTY, M.D.
SAN FRANCISCO

I shall show by a series of statistics why my improved Thiersch graft is superior to all forms of tampon, in producing better hearing and in shortening the time of treatment as well as in being more comfortable for the patient. I shall also demonstrate from my own cases that reoperation is not at all necessary provided the work is thoroughly done.

The reason for the improved hearing is easily understood when it is remembered that the graft is applied at the time of the first operation. In about eight or twelve days the graft will be loose and should not be lifted away until it is, because granulations will develop, the granulating surfaces producing a mechanical hindrance which impairs the hearing; while if the graft is lifted off and the underlying surface does not bleed, you can feel assured beyond a question of doubt that the new

TABLE 1.—EIGHTEEN CASES OF RADICAL EAR OPERATION BY IMPROVED THIERSCH GRAFT

Case No.	Age	Sex	Discharge	Operative Findings	Hearing Before and After Operation		Duration of After-Treatment (Weeks)*
					Before	After	
104	26	F.	Childhood.	Cholesteatoma.	Whisper, 6 ins.	Whisper, 6 ft.	Six.
105	14	M.	Two years.	Cholesteatoma.	Speech, water, 6 ft.	Speech, water, 15 ft.	Four.
107	28	M.	Childhood.	Cholesteatoma.	Whisper, 3 ft.	Whisper, 3 ft.	Six.
108	19	F.	Childhood.	Cholesteatoma.	Whis'r, water, 12 ft.	Whis'r, water, 26 ft.	Four.
109	10	M.	Childhood.	Cholesteatoma.	Whisper, 1 ft.	Whisper, 26 ft.	Four.
110	14	M.	Childhood.	Cholesteatoma.	Whisper, 1 ft.	Whisper, 25 ft.	Four.
111	25	M.	Two years.	Cholesteatoma.	Whis'r, paper, 3 in.	Whisper, 6 ft.	Six.
113	33	F.	Childhood.	Cholesteatoma.	Whis'r, paper, 3 ft.	Whis'r, paper, 20 ft.	Three.
115	26	M.	One year.	Cholesteatoma.	Whis'r, paper, 15 ft.	Whis'r, paper, 26 ft.	Four.
117	16	M.	Childhood.	Caries.	Whis'r, paper, 1 ft.	Whis'r, paper, 6 ft.	Four.
118	12	M.	Five years.	Caries.	Speech, 3 ft.	Whis'r, chair, 25 ft.	Three.
119	16	M.	Two years.	Caries.	Whisper, 25 ft.	Whisper, 3 ft.	Four.
120	9	M.	Seven years.	Cholesteatoma.	Whisper, 1 ft.	Whisper, 6 ft.	Three.
121	29	F.	Childhood.	Caries.	Whisper, 6 in.	Speech, 1 ft.	Three.
122	29	F.	Childhood.	Caries.	Whisper, 3 ft.	Speech, 3 ft.	Four.
123	4	F.	Three and one-half years.	Cholesteatoma.	Not made.	Good.	Four.
124	30	M.	Childhood.	Cholesteatoma.	Whis'r, chair, 2 ft.	Whis'r, chair, 15 ft.	Five.
125	18	F.	Ten years.	Cholesteatoma.	Speech, 2 ft.	Whisper, 25 ft.	Four.

SUMMARY

Duration of after-treatment:

In 4 cases 3 weeks. In 1 case 5 weeks.
In 10 cases 4 weeks. In 3 cases 6 weeks.

Hearing, following operation, for whisper:

In 6 cases 25 feet or more. In 2 remained the same.
In 3 cases 15 feet or more. In 1 case could not be measured, but good.
In 3 cases 6 feet. Three cases do not hear so well because of complications developed before or after operation.

Improved 14, or 78 per cent. Unchanged, 2, or 11 per cent. Made worse, 16 per cent.

* The duration of the after-treatment dates from the day of the radical mastoid operation and not from the day of the graft, which was four days later.

TABLE 2.—TWELVE CASES OF RADICAL EAR OPERATION BY IMPROVED THIERSCH GRAFT

Case No.	Age	Sex	Duration	Operative Findings	Hearing		Duration of After-Treatment (Weeks)
					Before	After	
127	21	M.	Childhood.	Caries.	Whisper.	Improved.	Three.
128	21	M.	Childhood.	Caries.	Whisper, 3 ft.	Improved.	Two.
129	31	M.	Childhood.	Caries.	Whisper, 6 in.	Whisper, 25 ft.	Ten.
130	38	M.	Five years.	Caries.	Whisper, 6 in.	Whisper, 18 ft.	Six.
131	20	F.	Childhood.	Caries.	Whisper, 3 in.	Remnants.	Four.
132	46	M.	Childhood.	Caries.	Whisper, 2 in.	Whisper, 15 ft.	Four.
133	34	F.	Fourteen years.	Negative.	Whisper, 3 ft.	Whisper, 26 ft.	Three and one-half.
134	17	M.	Eight years.	Caries.	Whisper, 6 in.	Whisper, 6 ft.	Three and one-quar.
135	9	M.	Three years	Caries.	Whisper, 1 in.	Whisper, 4 ft.	Three and one-half.
136	7	F.	Infancy.	Caries.	Whisper, 3 ft.	Whisper, 25 ft.	Four.
137	17	M.	One year.	Caries.	Whisper, 6 ft.	Whisper, 26 ft.	Three.
138	9	M.	Childhood.	Caries.	Whisper, 3 ft.	Not made.	Three.

Improved 100 per cent. One with labyrinth destroyed, the other lost.

TABLE 3.—THIRTY-SIX CASES OF RADICAL EAR OPERATION BY IMPROVED THIERSCH GRAFT

Case No.	Age	Sex	Discharge	Operative Findings	Hearing Before and After Operation		Duration of After-Treatment (Weeks)
					Before	After	
139	28	M.	Childhood.	Caries.	Whisper, 12 ft.	26 ft.	Six
140	11	M.	Childhood.	Cholesteatoma.	Whisper, 3 ft.	25 ft.	Four
141	10	M.	Childhood.	Caries.	Whisper, 3 ft.	15 ft.	Four
142	19	F.	Eleven years.	Caries.	Speech, contact	Speech, 6 ft.	Six.
143	19	F.	.....	Caries.	Speech, 3 in.	Speech, 20 ft.	Three.
144	29	M.	Ten years.	Caries.	Speech, 1 ft.	Speech, 6 ft.	Not cured.
155	29	M.	Eleven years.	Caries.	Whisper, 6 ft.	Whisper, 12 ft.	Four.
145	12	M.	Childhood.	Cholesteatoma.	Whisper, 3 ft.	Recovered; lost.	.....
146	20	F.	Childhood.	Caries.	Whisper, 6 ft.	Whisper, 26 ft.	Five.
147	65	F.	Childhood.	Cholesteatoma Labyrinth operation.	.....	.....	.....
148	39	F.	Childhood.	Cholesteatoma.	Whisper, 2 ft.	Speech, 6 ft.	Six.
149	7	M.	Three years.	Cholesteatoma.	Whisper, 3 ft.	Speech, 6 ft.	Six.
150	12	M.	.....	Caries.	Whisper, 6 in.	Whisper, 10 ft.	Six.
151	56	F.	Childhood.	Cholesteatoma.	Does not hear; labyrinth operation.	.....	.....
152	19	M.	Childhood.	Cholesteatoma.	Speech, 1 ft.	Speech, 1 ft.	Six.
153½	20	M.	Two years.	.....	Whisper, contact.	Whisper, contact.	Four.
154	33	M.	Childhood.	Caries.	Whisper, 3 ft.	Whisper, 3 ft.	Six.
153	33	M.	.....	Gun-shot wound of ear; removed bullet from brain.			
156	History lost		.....	.....	.....	.....	.....
157	17	F.	Infancy.	Caries.	Whisper, 1 ft.	Whisper, 20 ft.	Five.
158	10	F.	Infancy.	Cholesteatoma.	Whisper, contact.	Whisper, 2 ft.	Five.
158½	49	F.	Childhood.	Caries.	Whisper, 3 in.	Whisper, 15 ft.	Five.
159	19	M.	Childhood.	Cholesteatoma.	Whisper, 1 ft.	Whisper, 15 ft.	Six.
160	10	F.	Infancy.	Cholesteatoma.	Whisper, contact.	Whisper, 5 ft.	Four.
161	21	M.	Six years.	Caries.	Whisper, 3 ft.	Whisper, 15 ft.	Six.
162	23	M.	Childhood.	Caries.	Whisper, 1 ft.	Whisper, 1 ft. Sinus thrombosis.	Ten.
163	49	F.	Childhood.	Caries.	Whisper, 3 in.	Whisper, 3 ft.	Four.
164	52	M.	Childhood.	Cholesteatoma.	Comatose.	Purulent meningitis.	.....
165	9	M.	Childhood.	Caries.	Whisper, 3 in.	Whisper, 3 ft.	Five.
166	9	M.	Childhood.	Caries.	Whisper, 6 in.	Whisper, 6 ft.	Eight
167	34	F.	Childhood.	Caries.	Whisper, 3 ft.	Whisper, 20 ft.	Four.
168	37	F.	Twenty years.	Caries.	Whisper, 5 ft.	Whisper, 5 ft.	Four.
169	42	M.	Childhood.	Cholesteatoma.	No hearing, acute laby.	Whisper, 6 in.	Five.
170	12	M.	Two years.	Cholesteatoma.	Whisper, 20 ft.	Whisper, 6 ft.	.....
171	3	M.	Two years.	Caries.	Not reliable.	.....	.....
172	14	M.	Childhood.	Cholesteatoma.	?	?	?

epidermis will develop in a very few days, having at first a pinkish appearance which gradually fades to white. What more could be wished for than the internal ear covered by a single layer of epidermis? Can you in your fondest imagination picture granulation tissue covered with epidermis that is not more of a hindrance to the sound waves than this single layer?

Should grafts be applied at a second operation, they will all remain and become firmly attached if the improved technic is used.

I believe that the granulation tissue which remains after a thorough curettement is also a hindrance—macroscopically it may be removed, but a something remains

to fasten the graft and keep it in place; with the other procedure, a single layer covers the internal ear.

In a series of fifty-three selected cases, scarcely one required more than six weeks for complete epidermization. This result speaks for itself. This method lessens the likelihood of new caries developing, as it often does, in an ear that is tamponed because of the intense congestion and pus-formation over a long period of time.

In my own series of sixty-six cases in which I have applied the improved Thiersch graft it was necessary to reoperate in but one, and the patient was entirely free from secretion in four or five weeks after the second operation.

I believe that there is more likelihood of successful results from an incomplete operation by the improved Thiersch graft than by any other form of operative procedure because of the fact that the epidermis is so very thin that it will follow and cover individual cells that were not noticed at operation, and provided a large pathologic lesion is not left, it will correct itself or show as a small granulating surface that can easily be curetted with the sharp spoon as soon as it appears. It must be borne in mind that there is no granulating surface with the improved Thiersch graft and as soon as a granulation appears, it must be curetted away. I have found that such a spot packed with gauze saturated with sterile liquid petrolatum for twenty-four hours following the curettement will be much improved the following day and the place that was curetted can easily be detected.

SUMMARY

Male 21, female 14; lost history, 1.
Age 3 to 10, 7 cases; age 10 to 20, 10 cases; age 20 to 30, 7 cases; age 30 to 40, 5 cases; age 40 to 50, 5 cases; age 50 to 60, 1 case; age 60 to 70, 1 case.

The length of time required for complete epidermization was, in a series of twenty-nine cases without cerebral complications, the following:

In 3 cases 3 weeks.
In 15 cases 4 weeks.
In 6 cases 5 weeks.
In 4 cases 6 weeks.
In 1 case 7 weeks.

In this series of thirty-six cases there were the following brain complications:

1 case of gunshot wound of the ear and brain abscess; operation and recovery.
1 case acute purulent meningitis; operation, death.
1 case sinus thrombosis; operation, recovery.
2 cases acute labyrinthitis; no operation, recovery.
2 cases purulent labyrinthitis; operation, recovery.

In this series, there were nine cases of reoperation, five of my own; two patients did not recover from the acute operation and three from the radical. Two of these radical operations had been done prior to five years before. Four had been done by other operators.

In one of the purulent-labyrinth cases I had operated myself some five years previously but discharge continued.

The seven cerebral cases should not be included; detailed histories of two were lost; one of a child is not reliable, leaving twenty-five cases that can be concluded reliable.

Improved, 19 cases.....76 per cent.
Remained same, 4 cases.....16 per cent.
Made worse, 2 cases..... 8 per cent.

I have previously reported[1] twelve cases in which the improved Thiersch graft was used, two of which should be excluded; in every one of the remaining ten cases—100 per cent.—there was improvement. I have also reported[2] (see Table 1) eighteen cases in which the improved Thiersch graft was used:

13 cases improved.....72 per cent.
2 cases remained the same.....11 per cent.
3 cases were made worse.....17 per cent.

Adding my selected cases, I have fifty-three—

42 cases improved.....80 per cent.
6 cases remained the same.....11 per cent.
5 cases made worse..... 9 per cent.

I have reported[3] 100 cases of radical mastoid by tampon and Thiersch graft combined. In the latter thirty of the 100 grafts were used. In some instances they were good, in others they came away with the dressing. It was at this time that I first began the oil dressings.

In eighty-nine cases selected—

57 cases improved.....64 per cent.
26 cases remained the same.....30 per cent.
6 cases made worse..... 7 per cent.

The added improvement in this series of cases in which operation was performed was dependent on grafts.

Dr. Buhe[4] found that out of 100 cases which he examined at the clinic at Halle, 34 per cent. improved, 36 per cent. remained stationary and 30 per cent. became worse after the radical operation. Improvement or no change of hearing is to be found practically without exception if the labyrinth and labyrinth wall are intact and the distance of hearing is under a meter for whispering speech.

Improvement may be expected even if the labyrinth or labyrinth wall or both are affected, if the remnant of hearing extends over less than one quarter of a meter. On the other hand, a diminution of hearing sets in in the case of patients who heard for the distance of 3 feet or more before operation if the labyrinth was intact and remained so. The hearing diminishes also, if the labyrinth or labyrinth wall or both are affected and the hearing extends over more than a quarter of a meter.

A. Schonemann[5] says that in general the hearing is not affected by the radical operation if the labyrinth was previously intact and was not damaged during the operation. Hammerschlag[6] agrees in general with Wagner's statements.

Out of thirty-three cases (Hammerschlag's):

7 improved.....22 per cent.
3 made worse..... 1 per cent.
23 unaltered.....70 per cent.

Grunert[7] reports:

39 improved.....57 per cent.
4 made worse.....0.5 per cent.
28 unaltered.....40 per cent.

Grossmann[8] reports out of 212 cases:

93 improved.....44 per cent.
51 made worse.....25 per cent.
68 unaltered.....33 per cent.

1. Welty, C. F.: Improved Technic of the Thiersch Graft Following Radical Ear Operation, THE JOURNAL A. M. A., Sept. 16, 1911, p. 962.

2. Welty, C. F.: Ann. Otol., Rhinol. and Laryngol., March, 1911; Arch. internat. de laryng., January and February, 1911, p. 70; Arch. f. Ohrenh., lxxxv; Jour. Laryngol., London, June, 1911.
3. Welty, C. F.: California State Jour. Med., 1910.
4. Buhe, E.: Verhandl. d. deutsch. Gesellsch. Naturforscher u. Aerzte, 74 Versammlung, Karlsbad, 1902, II, 2, 44.
5. Schonemann, A.: Ueber den Einfluss der Radikaloperation am Gehörorgan auf das Hörvermögen, Cor.-Bl. f. schweiz. Aerzte, 1906, xxxvi, 458.
6. Hammerschlag: Wien. klin. Wchnschr., 1899.
7. Grunert: Arch. f. Ohrenh., xl.
8. Grossmann: Arch. f. Ohrenh., lii.

Gompertz[9] reports of eight cases:

4 improved.....50 per cent.
2 made worse.....25 per cent.
2 unaltered.....25 per cent.

Records are found of fifteen cases of A. von Ruppert's,[10] out of which there were:

7 unaltered.....47 per cent.
7 improved.....47 per cent.
1 made worse..... 6 per cent.

Brown[11] reports that in the cases in which perception of high notes was shortened before the operation and in which vestibulary symptoms had been found previously, after the operation a considerable and sometimes even a complete loss of hearing took place. This also occurred in cases with a normal perception of high notes, but which presented pathologic increase or diminution of vestibulary reaction. Out of these cases in which the hearing distance was from 0 to 5 meters, before operation:

30 per cent. showed improvement.
10 per cent. remained unaltered.
6 per cent. became worse after healing.

Of those cases in which the hearing distance was from 6 to 9 meters:

50 per cent. improved.
50 per cent. became worse.

When the hearing distance was 10 meters or more:

14 per cent. improved.
14 per cent. remained unaltered.
72 per cent. became worse.

In general, therefore, we may say that the hearing is reduced after operation in the majority of cases; an improvement in hearing, however, might be expected in cases in which the hearing was considerably diminished before the operation took place.

C. A. Torrigiani[12] says that the hearing will be the better, the more conservative the operation. If the labyrinth is intact, the hearing will be better or unaltered; but if the labyrinth is affected, the hearing will be unaltered or worse. Shortening of the bone conduction gives a bad prognosis so far as the hearing function goes.

E. Ruttin[13] says that out of 100 cases the hearing distance in forty cases was, before operation, from 1.5 to 2 meters for conversation voice. In eight of these cases the hearing improved after the operation. In six it remained stationary, and in twenty-six it diminished. Two of the cases which improved, showed regeneration of the mucous lining in the tympanic cavity which was persistent after healing.

Out of sixty cases in which there was a hearing distance of 1.5 meters or less, only three showed any improvement after the operation and two became worse. (In these statistics those cases are not included which had previously shown any labyrinth symptoms.) We can, therefore, say that in cases in which the hearing distance is from 1.5 to 2 meters, a diminution of the hearing takes place, especially if only a small perforation existed before the operation was performed. In cases in which the hearing distance is less than 1.5 or 2 meters, no considerable diminution is to be remarked.

In the eight cases showing improvement and in which the hearing distance was over 1.5 or 2 meters, the hearing distance increased from:

4 to 6 meters
4 to 7 meters
3 to 7 meters
4 to 6 meters
4 to 7 meters
4 to 7 meters
4 to 7 meters

In those cases in which the hearing distance is less than 1.5 meters the improvement in three instances was from

1.5 to 6 meters
10 cm. to 1.5 meters
1.25 to 3 meters

Trautmann and Stenger (quoted from Ruttin) report out of sixty-nine cases:

25 improved.....36 per cent.
20 diminished.....30 per cent.
24 unaltered.....35 per cent.

Schwartze (quoted from Ruttin) out of twenty-seven cases:

9 improved.....33 per cent.
14 unaltered.....52 per cent.
4 improved.....15 per cent.

Brieger (quoted from Ruttin) reports: 43 per cent. of improvements, 8 per cent. made worse and 49 per cent. unaltered.

Stacke (quoted from Ruttin) reports 35.3 per cent. improved, 7.1 per cent. made worse and 57 per cent. unaltered.

Manasse and Wintermantel (quoted from Ruttin) report thirteen cases, or 65 per cent. improved, one case, or 2.5 per cent. diminished and six, or 30 per cent. unaltered.

Siebenmann (quoted from Ruttin) reports sixty-one improved, or 61 per cent.; eleven improved, or 11 per cent. and twenty-eight unaltered, or 28 per cent.

It goes without saying that bone conduction will be benefited only to the extent that the mechanical hindrance is removed and that mechanical hindrance can be judged somewhat by the appearance of the tympanic cavity. While with a lengthened or normal bone conduction, you can promise an improvement in hearing provided that the patient does not hear a whisper from a distance of more than 6 or 9 feet. This is the rule I follow myself and advise my patients accordingly.

In all patients under 50 years of age who hear a whisper from a distance of 5 feet or less, you can promise that the hearing will be improved, unless something goes seriously wrong with the operative procedure; for instance, removing the graft too soon (so that granulation tissue takes its place), or allowing it to remain permanently, in which case it will act as a mechanical hindrance. In patients who hear a whisper from a distance of 10 or 15 feet, I do not promise any improvement. In patients who hear a whisper from a distance of 20 feet or more, I say that hearing will be improved.

In fact I am so enthusiastic on this particular point that I am desirous of securing a few cases of chronic adhesive processes in order to do the radical ear operation with the improved Thiersch graft. Provided the patients have an intact labyrinth, and the hearing for a whisper is below 10 feet, I am almost certain of improvement. The radical ear operation was done some eighteen years ago by a French surgeon for this purpose.

9. Gompertz: Monatschr. f. Ohrenh., 1902.
10. von Ruppert, A.: Ztschr. f. Ohrenh., liv.
11. Brown: Arch. f. Ohrenh., lxxx.
12. Torrigiani, C. A.: Arch. di Otologia, 1911.
13. Ruttin, E.: Ann. d. mal. de l'oreille, 1908, xxxiv, 391.

V. 191

It was abandoned because the result was not so good as wished for.

By removing the mechanical hindrance of the organized connective tissue, I can see a great advance in the field of otology as well as another indication for the radical ear operation.

I cannot imagine a more ideal surgical procedure for the improvement of hearing in chronic suppurative otitis media than this operation coupled with the improved Thiersch graft as my results speak for themselves; moreover, many otologists from the West can testify to the same. The time of the after-treatment is much shortened and is not at all painful.

In fact, the radical ear operation has lost its interest for me because it ceases to be a speculative procedure.

Shreve Building.

ABSTRACT OF DISCUSSION

DR. WILLIAM L. BALLENGER, Chicago: I wish to confirm some of Dr. Welty's conclusions. If we operate early and granulations follow, cicatricial contractions are likely to be followed by impaired hearing. Ten or fifteen years ago when we used the Thiersch's grafting, I used the oil dressing; for fifteen years I used the oil dressing. I was much interested in his statement that these granulations do not follow the use of the improved Thiersch graft, but that so soon as they do appear they should be corrected. If they do not appear, why is it necessary to correct them?

I should like to know what tests were made after the operation. After one or two years, or even three years, many of these cases will show a great improvement in hearing because of cicatricial formation. Again, one who can hear a whisper at 20 feet is made worse by the radical operation. I should not object to doing anything except the modified radical operation. I have done the modified operation for five or six years in those cases which did not show any marked impairment in hearing, and almost universally there has been a marked improvement of hearing. In many cases the improvement is simply marvelous. I should do the modified radical operation in many of the cases in which Dr. Welty does the radical operation.

DR. J. HOLINGER, Chicago: Microscopic examinations of chips of bone containing so-called cholesteatoma membrane show that this membrane is simply a scar over the bone. A layer of hornified epidermis-cells covers the bone and there is very little connective tissue between the bone and the layer of hornified epithelial cells. Often you will find under this layer of epidermis giant cells in typical Howship's lacunae. They indicate the process which is the cause of the cavities enlarging automatically. Dr. Welty spoke about improvement in hearing. If you do any promiscuous scraping you may cause hemorrhage and you are liable to destroy hearing by injuring the stirrup. Therefore, rather protect the little islands of epidermis. I cannot see how the transplantation of epidermis into the mastoid cavity will cause an improvement in hearing. The main point is to leave the stirrup alone. The incus and malleus may be necrotic and have to be removed, but then hearing is not normal. I started to make Thiersch grafts in 1893 and have used them for many years. In 1898 I showed a number of cases before the Chicago Medical Society which had remained dry for three and four years. Since that time I have used various other means to epidermize cavities of radical mastoid operations. I have discarded the Thiersch grafts and use other methods which are less troublesome to the patient and to me, and I obtain better and quicker results. I have often seen dry cavities after from three and one-half to four or five weeks. It seldom takes six weeks to get a result.

DR. J. C. BECK, Chicago: I shall take up the subject where Dr. Holinger left off. In the reformed membrane there is epithelial tissue but no granulation tissue. It stands to reason that between the bone and the epidermis there must be some connective tissue framework. I have demonstrated to my own satisfaction the pathologic condition of the bone operated on. There are six different types of cases: (1) osteosclerosis, (2) osteofibrosis, (3) osteocholesteatoma, (4) tuberculosis, (5) osteofibrosis with syphilis and (6) reduction of the sequestra after going on to the chronic form.

One man may report his treatment successful and another man may report his treatment successful, although the treatments differ. I am sure that the use of the Thiersch graft, after the thorough removal of the diseased process, will result favorably. The prompt application of the Thiersch graft hastens recovery, and this is very important.

There is a prompt and lasting improvement in the hearing if the diseased process is thoroughly removed and the Thiersch grafting done promptly.

DR. CULLEN F. WELTY, San Francisco: To those of you who have not used the oil dressing, I can recommend it in the strongest terms. You can assure your patients that they will be well in six weeks or less. In this procedure the detailed technic must be followed to produce satisfactory results. The most important part of this paper has not been discussed at all and that is the amount of hearing following operation. The reason for this improvement is quite obvious when you consider the healing process. Such a series of cases has never been presented before, and this is all due to the graft applied by my method. I have patients who continue to hear a whisper at a distance of 25 feet after three years. I am more enthusiastic than ever in regard to operative treatment in chronic suppurative otitis media.

DIFFERENTIAL DIAGNOSIS AND TREATMENT OF CHRONIC LARYNGITIS

EMIL MAYER, M.D.
NEW YORK

As functional laryngitis is to be the subject of another part of this symposium, the differential diagnosis and treatment of organic laryngitis only is here presented. It is to be differentiated from affections of the larynx caused by constitutional affections such as syphilis, tuberculosis, lupus aunl scleroma; from the benign laryngeal growths, papilloma, mucocele, etc.; from malignant growths which so frequently involve the larynx; from prolapse of the ventricle; from lesions of the mucous membrane allied to affections of the skin as pemphigus, and from lesions caused by interference with the nerve supply of the larynx, the abductor and adductor paralyses.

It is not necessary here to go into detail of the differential diagnosis. The mere mention of the various ailments that may similate chronic laryngitis is quite sufficient to indicate what we have to bear in mind and against which we have to guard in making our diagnosis.

There is always a preceding acute inflammation, although the interval between the acute and chronic condition may be very short.

The treatment of this affection cannot be successful unless the etiologic factors are fully known. Of these first in line come the obstructive conditions of the nose, occasioning mouth breathing and preventing properly warmed air from entering the larynx.

Chronic catarrhal conditions of the nose and nasopharynx extend into the larynx by contiguity and produce a chronic laryngitis. Treatment must be vigorously applied to the nose and nasopharynx before amelioration of the laryngeal symptoms can occur.

One of the most frequent causes of laryngeal irritation and chronic inflammation is either the elongated uvula or its papillomatous tip. Simple as it may seem,

it is surprising how frequently this condition is overlooked, as a single case in point may well illustrate.

A young woman, aged 20, came to me because of cough and constant clearing of the larynx, lasting for the past eighteen months, during which time she had been receiving treatment of all kinds of applications without benefit. I found the laryngeal mucosa very much engorged and by carefully elongating the uvula in depressing the tongue, found a long papillomatous tip which ordinarily curled up behind the uvula as it retracted on examination. Uvulotomy was followed by complete cessation of all the symptoms.

Hypertrophied lingual tonsils cause laryngeal irritation and require removal or galvanocautery applications before any direct treatment to the larynx.

Follicular pharyngitis may keep up a laryngeal irritation. For the rapid removal of these I have devised a curet.

Pressure in the auditory canal is another cause of laryngeal irritation. I have recorded a case of the cure of a constant cough and rasping by the removal of a plug of cotton which had been placed in the ear six months previous to the examination and forgotten.

Constitutional disturbances, digestive and cardiac, and pressure of new growths and enlarged glands are causative factors in producing chronic laryngitis, and the relief of those conditions, where possible, means ability to cure the laryngeal disturbances. Chronic laryngitis may be an occupational disease following irritation from gases or a dust-laden atmosphere, and in these the wearing of a respirator is a necessity as a preventive.

Alcoholics are especially susceptible, as also are males at the age of puberty. The use of tobacco is a causal factor and its continued use often retards a cure.

Sudden changes of temperature are most common causes of laryngitis.

Inflammatory conditions of the larynx occur from improper use of the voice in public speakers, actors and singers, and these are the patients who tax our capabilities to the utmost, for impairment of voice to them means a financial loss, often great. Their disturbances may range from a slight loss of tone in the upper register, which can only be noted by an expert, to a total loss of voice. These patients must be treated with the utmost care and firmness, as so much depends on their recovery, and harm may be done by over zealous treatment. One should hesitate before performing any operation on the nose or throat of any patient of this class without most mature consideration.

It must be borne in mind that the singer has learned his art with the very pathologic condition that we have before us and its removal may disturb the equilibrium of his vocalization, and should the laryngitis still continue, the failure to return to normal will be ascribed to the operative interference.

The conditions here are usually local hypertrophies, such as the singer's nodes, which are limited to a characteristic region, namely, the junction of the anterior third with the posterior two-thirds of the vocal cords and occur as a result of friction.

After the removal of any existing causal factor, our first order must be: Rest. The more complete, the better.

Local applications may be made of a spray of the tincture of chlorid of iron, half a dram to the ounce of water. In the more chronic conditions the following solution is given:

		gm.	or c.c.	
℞	Iodin	1		grs. viii
	Potassium iodid	2		grs. xvi
	Glycerin			
	Water ..... āā	90		℥ lss

Chlorid of zinc and solution of alum, 30 grains to the ounce, are also used, applied locally. During the day medicated lozenges containing benzoic acid or krameria are beneficial. Steam inhalations, specially with the resinous substances such as are contained in the compound tincture of benzoin, are especially soothing and relieve much of the irritation, but far better results are obtained if the medicated steam is applied methodically and regularly, as may be done in well-equipped inhalatoria, and these should always be under the direction of a competent physician. When this is not obtainable, special inhalation apparatus may be obtained, and this form of treatment may be carried out under the physician's personal supervision.

There are two methods of inhaling; in the first the medicament is atomized to impalpable vapor by means of sterilized air in a cabinet, while in the second the inhalation is through a sterile mouth-piece or nose-piece.

The medicated steam, a mixture of compound tincture of benzoin, one in twenty, is inhaled at a temperature of from 140 to 170 F. for about fifteen minutes. This is followed by inhalations of oily substances without the aid of heat, either of the two following being used:

		gm.	or c.c.	
℞	Camphor	5		ʒ i
	Oil of tar	10		ʒ ii
	Iodin	2		gr. xx
	Creosote	5		ʒ i
	Menthol	5		gr. xxx
	Oil of sesame	50		ʒ iv

or

a 5 per cent. solution of the following:

		gm	or c.c.	
℞	Iodin	5		ʒ vi
	Oleic acid	15		ʒ ii
	Paraffin oil	30		ʒ iv
	Oil of sesame, to make ..	500		oi

The patient remains indoors for from fifteen to thirty minutes after each treatment. The use of tobacco is prohibited.

Faulty methods in vocalization should be corrected. Often a complete change of air and surroundings is advisable, and a few days or longer at some seacoast resort under favorable weather conditions may be of great benefit.

Cold sponging of the upper part of the body, the wearing of proper clothing, regularity as to meals, regulation of the bowels should be part and parcel of the directions toward the cure and prevention of recurrence of chronic laryngitis.

40 East Forty-First Street.

No Justification for the Rat.—Of all the parasites that have their being in and around the habitation of man, the rat has less to justify its existence than any other. As devoid of any redeeming traits as the fly, which has been the subject of a nation-wide sanitary crusade, the rat is a greater pest because of its depredations and its possibilities for harm in the transmission and perpetuation of bubonic plague in any community. The latter consideration is of more serious import in seaport towns, wherever they may be, and in those localities where plague has once appeared, but with the world-wide march of bubonic plague, in no city should its advent be considered as improbable.—R. H. Creel, *Pub. Health Rep.*

V. 191

DIAGNOSIS AND TREATMENT OF SYPHILITIC LARYNGITIS

JOSEPH C. BECK, M.D.
CHICAGO

A remarkable change has taken place in the knowledge of syphilis in the past three years, in that the specific organism, the *Spirochaeta pallida*, has been positively identified as the etiologic factor, the serologic examination of the blood, the so-called Wassermann reaction, has been accepted as positive proof of the disease, and finally a great remedial agent has been added to our armamentarium in the form of an arsenical preparation, salvarsan, which has been demonstrated beyond question as the great sterilizer of the whole system of the spirochete. We are indebted primarily for this triad of discoveries to Professors Schaudinn, Wassermann and Ehrlich and their many earnest collaborators who have so dilligently taken up these modern thoughts and tried them out so as to put the management of this affection on a satisfactory basis. All text-books and articles that were published before these three discoveries of etiology, diagnosis and treatment may be properly called antiquated. One of the most recent articles and texts on this subject containing a wealth of information is by Felix Blumenfeld,[1] and I have made free use of it.

The pathologic pictures (Figs. 1 to 36) and the antiluetic treatment by mercury and potassium iodid, however, are still our mainstays, and very little new has been added in this particular. It is now a well-established fact that salvarsan alone is not a cure in the majority of the cases and mercury and potassium iodid are essential in the treatment. Syphilitic disease, confined purely to the larynx, is comparatively rare, and there are usually associated lesions of the nose, nasopharynx, mouth, tongue, and oropharynx. There are two principal forms of syphilitic laryngitis: (*a*) congenital and hereditaria tarda; (*b*) acquired.

The former type, namely, congenital syphilis, according to Karl Gerhard[2] is very rare and is rarely diagnosticated except post mortem. In the hereditaria tarda one finds associated with this affection the triads of Hutchinson and the predilection of attack is the epiglottis. The ultimate changes are usually very severe and great cicatrices are found requiring surgical intervention.

ACQUIRED FORMS

Of the acquired forms we must consider the following:

1. Primary chancer.—This is a great rarity, and only a few cases are on record. Morel Mackenzie, Moure, Pryat, Krishaber, and Castex have seen this condition and in all the cases the lesion was in the epiglottis. Sarremone also reports a primary chancer of the larynx, but the lesion was in the right ventricular band.

The syphilitic laryngitis, associated as the constitutional affection, may be classified as: (1) erythematous, (2) papular, (3) condylomatous, (4) diffuse infiltration, (5) nodular, (6) gummatous—diffuse gummatous infiltration, or solitary gummata, (7) perichondritis, which may either suppurate or not, and finally form cicatrices.

The erythema may well be compared to a catarrhal condition, only much deeper injection of the mucosa, and at times may assume an edematous character. Lacroix[3] described such a form of syphilitic laryngitis. The papules are also of the early form of luetic laryngitis. Condylomas are not very frequent and also belong to the very early manifestations, although Aronson[4] observed a case which occurred nine years after the primary infection. These condylomas are usually situated on the edges of the vocal cords and frequently symmetrically. The infiltrated form of syphilitic

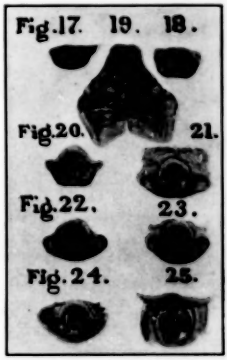

Fig. 1.—Erythema—small papule.
Fig. 2.—Papular forms.
Fig. 3.—Papular forms.
Fig. 4.—Papular forms.
Fig. 5.—Diffuse infiltrations with ulcerations.
Fig. 6.—Diffuse infiltrations with ulcerations.
Fig. 7.—Diffuse infiltrations with ulcerations.
Fig. 8.—Diffuse infiltrations with ulcerations.
Fig. 9.—Marginal ulcerations.
Fig. 10.—Subglottic ulceration and infiltration.
Fig. 11.—Unilateral extensive ulcerations.
Fig. 12.—Bilateral infiltration, edema and ulceration.
Fig. 13.—Gummatous formation, especially of the epiglottis and arytenoids, with breaking-down ulcerations.
Fig. 14.—Bilateral gumma of the arytenoids, without ulceration.
Fig. 15.—Marked destruction by ulceration of the entire epiglottis and one arytenoid.
Fig. 16.—Cicatrization of similar processes as in Figure 15.
Figs. 17-25.—Infiltration with ulceration and perichondritic changes.

1. Blumenfeld, F.: Handbuch der speziellen Chirurgie des Ohres und der obern Heftwege, iv, 375.
2. Gerhard, Karl: Haymann's Handbuch, i, No. 2, p. 1201.
3. Lacroix: Arch. internat. de laryng., No. 6, 1897.
4. Aronson: Arch. f. Laryng., xxii, 92.

laryngitis may be properly called the transitional stage from the early to the later form. The epiglottis is the most frequent location. The other regions of the larynx, however, are not infrequently infiltrated. These infiltrates may remain for a longer or shorter time before ulceration, but these defects are usually more superficial than the gummatous defects, and perichondritis is less frequent as a complication. The ulcerations are characterized by their sharp borders.

The nodular form of laryngeal syphilis is somewhat rare and bears a resemblance to lupus of the larynx. Lewin, who first described this affection, noted the rapidity with which these nodules broke down.

Solitary gumma of the larynx is more frequently present than it is diagnosticated and is frequently mistaken for other neoplasms. They vary in size from that of a pea to that of a walnut. It is usually deep red in color until the breaking-down process begins, when the color appears lighter.

The diffuse gumma of the larynx has a predilection for the epiglottis and not infrequently is the neighboring portion of the base of the larynx involved in the process. Frequently also the entire epiglottis is

Fig. 26.—Gumma of the epiglottis, with subglottic ulceration. Figs. 27 and 28.—Ulceration associated with cicatrization.

destroyed by the breaking down of the gumma. The epiglottis is often drawn to one side or the other, and in the final process becomes attached to the arytenoid so as markedly to obstruct breathing. When a diffuse gumma involves the cord, it is of a spindle form and of a very deep red color and when it breaks down has ragged edges.

In the perichondritic form of laryngeal syphilis it is the arytenoid that is most frequently involved; it is a late secondary process. There is usually median placement of the cord and the arytenoid is fixed. In the breaking-down process or abscess formation, the arytenoid has a tendency to lean forward and inward, and since the ary-epiglottidean fold is usually very much swollen or edematous, one can expect considerable difficulty with breathing and be on the alert for surgical interference. In case of suppuration of this perichondrial process it may come to the spontaneous expulsion of the arytenoid cartilages by coughing. The next in frequency of involved cartilage in luetic perichondritis is the cricoid. This causes considerable difficulty in swallowing, as the posterior laryngeal wall is principally swollen. Should the anterior cartilaginous arc of the cricoid become involved with perichondritis, then great difficulty in breathing will result—in fact, there is danger of suffocation. One may recognize this form of perichondritis, as also that of the thyroid cartilage, by a subglottic swelling and deep red color, as well as a swelling which sometimes crepitates externally over these cartilages. Not infrequently is there fluctuation present, which is due to the presence of an abscess.

The end-results from all the forms of destructive laryngeal syphilis, but especially those of the perichondritis, will either be the formation of granulation tissue in the form of excrescences or cicatricial bands. Both of these will cause more or less dyspnea.

A form of laryngeal syphilis in which there are no evidences of being or having been any ulceration is known as parasyphilis of the larynx and is characterized by general cicatricial narrowing of the cavity of the larynx. Dittrich[5] and Turck[6] have already described such conditions under different titles, and in recent years a number of cases have been recorded. Especially of great interest is the report of Gerber of such a case in which salvarsan had such splendid effect, whereas other antiluetic measures had no effect.

Figs. 29-36.—Cicatricial formations.

Rerol[7] examined a case of this sort microscopically and demonstrated that most probably the process originated submucously.

TREATMENT

The most important, in fact the only rational treatment, is by means of the antispecifics, namely, mercury, especially in the early stages, and potassium iodid in the gummatous and later stages. It is not within the province of this paper to go into detail of the general treatment; besides, almost everyone is familiar with it. There is, however, something to be said in reference to the use of salvarsan in the affection of the larynx. In the very early forms of syphilitic laryngitis it has been found that the most brilliant results were obtained by giving salvarsan intravenously in large doses, and thus preventing further progress by further amplifying the treatment with mercury. The same is true of the early ulceration and great swelling of the mucous membrane where suppuration is threatening. This treatment was at first employed with great trepidation, because it was feared that there might result a swelling of the mucous membrane secondary to the use of salvarsan, the so-called Herxheimer reaction, which would necessitate a tracheotomy, but Gerger found in several cases that this was not so. I have had three such cases of early laryngeal syphilis with great obstruction to breathing, which responded most beautifully and without any untoward symptoms following the use of salvarsan. Again, as

5. Dittrich: Prager Vitelgahrsch., 1850.
6. Turck: In Atlas Taf., 23.
7. Rerol: Thèse de Lyon, 1905, p. 58.

V. 191

mentioned before, in the so-called parasyphilis of the larynx, or in those cases that do not respond to mercury and potassium iodid treatment, salvarsan often changes the condition so that mercury and potash do have beneficial effects. Since syphilitic laryngitis is usually not a painful affection, one need not employ any general remedies for relief. The local treatment may be divided into topical applications, semioperative and operative.

Beginning with the possibility of making a diagnosis of a primary chancre of the larynx, one may practice what the genito-urinary specialists have successfully done with chancre on the penis; this is to resect the chancre, especially if it be localized on the epiglottis. In the early stages of erythema, papules and condylomas, the local application of calomel or iodol insufflations are very agreeable, mild solutions of silver nitrate or argyrol, steam inhalation, and mild alkaline sprays, are also indicated, especially when the secretions are excessive. The rest to the voice is very beneficial. In the broken-down infiltrations or gummata, it is necessary frequently to cleanse the surfaces by laryngeal irrigations or swabs, followed by application of stronger solutions of silver nitrate or iodoglycerin solution. The same is true in the broken-down nodular and perichondritic type. When the concomitant edema is present then frequently multiple punctures of this edematous membrane give marked relief. In the severe forms of perichondritis with abscess formation pointing externally, an early incision externally will prevent further extension or greater embarrassment to breathing. In all forms of the later stages of laryngeal syphilis, when considerable swelling is present, one must be constantly watching for the possibility of having to perform a tracheotomy, and in very severe forms of dyspnea it is frequently better not to wait till the last moment, but do a tracheotomy beforehand.

The management of the end-results of the ulcerated forms of laryngeal syphilis will depend on the condition present. The direct method of laryngoscopy, especially by the aid of suspension, offers great aid in the treatment of multiform cicatricial deformities and obstructions. Incisions of bands and resections of cicatricial masses with subsequent dilatation, either by the old Schrotter's dilators, directly by Jackson's dilator or bougies, or the wearing of the new O'Dwyer tube, as recently recommended by Mayer and Jackson, will no doubt give very satisfactory results. If, however, the interior of the larynx be markedly obstructed, especially following severe forms of perichondritis, with abscess formation in which a greater portion of the cartilages have sloughed away, then a laryngostomy will give the only permanent relief.

DIAGNOSIS AND TREATMENT OF LARYNGEAL TUBERCULOSIS

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In discussing the subject of tuberculous laryngitis I take it that it is not expected that time shall be expended in dwelling on the more obvious and well-known forms of the affection, but that some of the things learned from experience in the earlier, less marked lesions would be more profitable.

Those practicing in a place like Colorado Springs where there are many consumptive patients, encounter more cases of larynx involvement than do those who work in communities in which consumptives are relatively few; and we thus have perhaps more opportunity to see and study these cases.

I think, however, that patients with pulmonary tuberculosis are not as a rule subjected to systematic laryngeal examination early enough or often enough even in health resorts and sanatoriums. If this is done, it results in the discovery of many cases of incipient disease of the larynx, and appropriate treatment may be instituted at a time when such treatment is most effectual. The doctrine that early diagnosis enhances the prospect of a favorable outcome of treatment is perhaps just as true in this disease as it is when we are dealing with lung tuberculosis.

DIAGNOSIS

At the outset, let us not forget that primary laryngeal tuberculosis is an exceedingly rare disease, and further that not every case of hoarseness or even laryngitis in a tuberculous patient is necessarily due to tuberculosis of the larynx. Tuberculous persons may have simple catarrhal laryngitis or syphilitic or cancerous disease of the larynx, and the differentiation is not always an easy matter, especially in the beginning.

Hoarseness, pain (spontaneous and on swallowing, and shooting up to the ears), infiltration and ulceration of a typical appearance are the classical signs and symptoms of tuberculous laryngitis, and need not be described here; but a patient may have the disease and not be hoarse, as for instance, in a case of slight ulceration of the epiglottis or some other part of the larynx not directly concerned in phonation. I do not refer to that peculiar quality of the voice which may be described as "weakness" (for lack of a better term) which practically all sufferers from pulmonary tuberculosis present. This is probably due to a lack of vigor of the muscles of phonation and is a part of the general muscular atony. It differs from the hoarseness of tuberculous laryngitis as much as does the raucous voice of a syphilitic larynx. I have in mind two cases, in one of which the lesion was confined to a small and atypical ulcer on the left wall of the interior of the larynx above the false cord; in the other, the left side of the tip of the epiglottis was ulcerated and a slight infiltration developed later. In the first case, absolutely no pain and only the slightest huskiness was complained of; in the second, no hoarseness but merely a sense of discomfort in the throat was observed.

Then the sign of pallor of the mucous membrane which is given in most text-books as one of the diagnostic features of laryngeal phthisis is from my observation a very unreliable index, for the color depends on the complexion of the patient and the hemoglobin content of the blood. Pallor has no significance in this connection unless localized in the throat. I am aware that this is not an original discovery of mine, but I cannot forget my disappointment and perplexity during my early experience in not finding it always.

A sign which has seemed to me to be so constantly present that I attach great importance to it is a thin line of mucopus which lies in the posterior commissure and extends up and over the top of the interarytenoid region. I have thought that this might be due to a localized inflammation or ulceration in the subglottic region. This is, of course, attended by other indications of laryngitis.

Redness of one cord, the appearance of the other remaining normal, is almost diagnostic of tuberculous laryngitis in a tuberculous person.

I have often noticed a slight infiltration of the epiglottis—not the characteristic enormous thickening, but

a sort of clumsy-looking, blunt-edged organ in a larynx showing distinct but early signs elsewhere—which I have suspected may be a true tuberculous infiltration, though it has not always developed into the characteristic type.

In the differential diagnosis we must consider simple catarrhal laryngitis, pachydermia, syphilis, carcinoma and possibly posticus paralysis, rheumatic arthritis of the crico-arteniod joint and laryngitis sicca.

As mentioned before, a tuberculous person may have a simple catarrhal laryngitis, and it may be impossible to say at first that it is not the beginning of a local tuberculous process. It is only by observing the further course of the disease as to whether or not the condition soon clears up that a correct opinion can be formed. Every affection of the larynx in a phthisical patient should be regarded with suspicion, and the case kept under close supervision. A laryngitis which does not clear up promptly should be regarded as probably tuberculous; later on the characteristic signs will be discovered in most cases.

A pachydermia of the posterior wall sometimes strikingly resembles the granulating edge of a tuberculous ulcer in this situation, and pachydermatous areas may also become infected with the Koch bacillus. The differentiation may be very difficult; but the finding of an ulcer either by inspection or by the presence of blood on a swab with which it has been wiped, together with the discovery of lung tuberculosis, will lead us along the right track. Typical tuberculosis in other parts may be required to establish a diagnosis.

In laryngitis sicca the crusts may adhere so tenaciously and look so much like a tuberculous deposit that it is not possible from appearances alone to make a diagnosis. Injection of warm water or oil and clearing of the larynx by coughing will nearly always remove them.

As between syphilis and tuberculosis the chances of error have been greatly reduced since the Wassermann reaction has been in vogue and salvarsan discovered. In this connection it should not be forgotten that a mixed lesion is present in many cases, and we should be on our guard in a case of either one that the presence of the other is not overlooked. I have notes of a case in which a diagnosis of tuberculous laryngitis was made in a woman with a history of pulmonary and hip-joint tuberculosis. Later a characteristic skin syphilide was discovered and under antiluetic treatment the larynx rapidly healed except in a comparatively small area, which remained obstinate. This was most probably a case of syphilis of the larynx on which a tuberculous infection was grafted. Moritz Schmidt reports a case in which tuberculosis, syphilis and carcinoma were all present in the larynx of the same patient.

The distinction between carcinoma and tuberculosis of the larynx, especially of the tumor form, offers difficulties at times. Lagging of the affected side on phonation in carcinoma, the appearance of the lesion sometimes, and the age of the patient help to differentiate it. A tuberculin test is of great value, particularly if the local reaction in the larynx is looked for. Microscopic examination of an extirpated piece is in most cases decisive.

Unilateral posticus paralysis occurring in a person with pulmonary tuberculosis might be mistaken for a tuberculous perichondritis of the arytenoid with ankylosis of the crico-arytenoid joint; but the tumefaction and infiltration of the tissues would be absent in the former affection.

Rheumatism of the crico-arytenoid joint is, in my experience, a rare occurrence, and would therefore not often be a factor in the differential diagnosis. Furthermore, in rheumatism we would expect to find other joints involved.

TREATMENT

I am a firm believer in the idea that laryngeal tuberculosis ought to be thoroughly and persistently treated. I have no patience with the view that it might just as well be left alone because occasionally a patient recovers spontaneously or because these patients are doomed any way. The results of Schmidt, Heryng and a host of others prove that much can be done by appropriate treatment to add brightness to the prognosis in these cases.

Of first importance in the treatment is the proper care of the lung condition, because it is almost axiomatic that with a progressive lung disease little improvement can be expected in the process in the larynx. Therefore appropriate climatic environment, and proper supervision of the patient's daily life, habits, food, sleep and rest are basic essentials, together with such other therapeutic measures as seem necessary. In this connection the care of the appetite and digestion is of supreme importance. It has been well said that one's chances for recovery depend on a sound stomach. Tuberculin in suitable cases is useful, although I have observed no direct beneficial effect on the throat from its use.

Cough is a source of much irritation to an inflamed larynx, and should be controlled as far as possible by the voluntary efforts of the patient and attention to the upper air-passages, and subdued, if necessary, by narcotics, such as heroin or codein; the deleterious effects of these drugs on the appetite and digestion must at the same time be kept constantly in mind.

Talking causes as much irritation to the larynx as coughing, and except in those cases in which the lesion is so situated as not to be affected by its movements when phonating, speech should, as a rule, be interdicted. There are patients, however, who seem not to be able to exist if they are put on a strict "silence cure." They are born with a "gift of gab," have no resources of entertainment within themselves and get blue and melancholy if kept away from associates. With such persons the physician must use sound discretion and permit a limited amount of speech—perhaps require them to converse in whispers. He must remember always that he is treating a human being and not regulating a piece of inanimate machinery.

The relief of painful swallowing is just as essential from the point of view of maintaining a high degree of nutrition. No matter how hungry the patient may be he is not going to take a sufficient quantity of food if the effort is accompanied by excruciating agony. The local application of analgesics such as orthoform or anesthesin, either in powder or emulsion, is very useful. When the pain arises from lesions below the epiglottis, alcohol injections of the superior laryngeal nerve in some cases give complete relief lasting sometimes for days. Pain due to ulceration of the epiglottis can be relieved by amputation of this structure as emphasized by Lockard.

A great number of remedies have been used as local applications for the cure of laryngitis, prominent among them being lactic acid, formaldehyd solution, trichloracetic acid, Lake's mixtures, etc. I suppose each of us tries out many of them and then settles down to a few which seem efficacious. For my part I have felt that I have had best results from the use of liquor formaldehydi and trichloracetic acid. The former is of most value in

V. 191

infiltrations, used in strength of from 3 to 10 per cent. and thoroughly rubbed in, while the latter is reserved for ulcerations, applied in saturated solution at intervals of from seven to ten days. Both of these drugs find their greatest field of usefulness in cases which are not suitable for surgical measures; that is, in patients with active tuberculosis who are running high temperatures, or who have extensive involvement of the larynx or those whose equilibrium is too much upset by the thought of "cutting." The application of sunlight or Roentgen rays directly into the larynx are measures with which I have not had experience, but which I do not believe offer much if any advantage over the local application of drugs. Freudenthal speaks rather enthusiastically of fulguration, but I have seen no reports of its effects other than his original article.

We now have to consider a branch of the therapy which I believe is in suitable cases the most valuable aid in combating this affection. I refer to surgical measures. Ever since Heryng and Moritz Schmidt, a quarter of a century ago, began advocating surgical treatment of laryngeal phthisis, there has been at no time a complete accord on this proposition; and yet those men consistently adhered to their contentions, and the results have justified their faith. I feel convinced from the experience I have had with it that in properly selected cases it is the one best method of local treatment. Let no one, however, regard this plan as being universally applicable in all cases. He who does so will inevitably come to grief. But given a case of not too extensive involvement, with a quiescent lung, with a temperature of not over 100—in short, a patient who exhibits a fair amount of resistance to the tuberculous invasion—and favorable results may be confidently expected together with a shortening of the time required to bring the results about. Isolated tuberculomas or vegetations, moderate infiltrations and ulcerations, especially in early cases, are particularly responsive to the curet, the laryngeal punch or the galvanocautery. I have many times seen a tuberculous infiltration and ulceration of the posterior wall, which had for weeks and months resisted the application of various pigments and escharotics, yield promptly to the use of the punch, followed by lactic or trichloracetic acid. The cautery point is of much service in massive infiltrations of the false cords when the use of the double curet is perhaps unsafe.

Surgery is sometimes indicated also as a palliative measure. I recall the case of a young girl who had extensive involvement of the larynx and whose dysphagia was so great that she refused absolutely to swallow anything, even water. She was doing badly in every way and seemed altogether an unsuitable case for surgical interference. The case was desperate, however, and with the hope of affording some relief, a quantity of the offending mass was removed. The result was most happy. The patient was able to swallow with comparative comfort, her general condition, in consequence of the improved nutrition, improved and, while the ultimate result of the case was never in doubt, the temporary relief was well worth while.

Many cases of laryngeal tuberculosis arise in far-advanced cases of phthisis in which the prognosis is absolutely bad; yet even in these unfortunate cases surgery has a place. For instance, in dysphagia from ulceration of the epiglottis, when amputation of this structure is often followed by the greatest relief. Lockard says that this operation is indicated in any case, however bad, simply as a method of relief from suffering.

Tracheotomy and laryngofissure have been advocated as curative measures, although they seem to be too heroic to be adopted in any but a small minority of cases. Still there can be no doubt that tracheotomy does often exert a favorable influence on the disease by putting the larynx at rest. As an illustration, I refer to the following case: I performed a tracheotomy four years ago on a young man who had for several years been suffering with lung tuberculosis and whose larynx was quite extensively involved. As a result of increasing and extreme stenosis the tracheal opening was necessary to prevent suffocation. Neither his regular attendant nor I expected him to live three months. He disappeared from observation shortly afterward and I thought he had gone to his reward. Much to my surprise he walked into my office two years later with his larynx perfectly healed and with a good but slightly husky voice. He is still alive and his larynx remains well, although he will in all probability eventually succumb to the disease in his chest, which is quite extensive.

CONCLUSIONS

1. Sufferers from tuberculosis should have their throats examined early and often, and persistent vigorous treatment should be instituted at the beginning of trouble in the larynx.

2. Proper attention to the general condition is one of the prime essentials.

3. Surgical intervention in appropriate cases offers by far the best prospect of permanent relief, and not infrequently is of much value as a palliative measure.

The Colchester.

DIAGNOSIS AND TREATMENT OF PARALYSIS OF THE VOCAL CORDS

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In the diagnosis of laryngeal paralysis, one must remember the normal functions of the muscles and their nerve-supply. There are four pairs of muscles on each side which act alike and one single muscle acting on both sides at once. These are supplied by two pairs of nerves, the superior and the inferior or recurrent laryngeal nerves. Remember, the superior laryngeal nerve supplies all of the motor muscles on the outside of the larynx, consisting of just one pair, and sends no motor filaments to any other muscle. It is the sensory nerve for the whole laryngeal mucosa. All of the other laryngeal muscles are supplied by the purely motor recurrent laryngeal nerves.

The external (cricothyroid) muscle, which is supplied by the superior laryngeal nerve, regulates the tension of the cords and it is very rarely paralyzed. Paralysis of sensation is always due to involvement of the same nerve. Nearly all cases of paralysis of the vocal cords, and every case in which the outward and inward movements of the cords are interfered with, fall within the domain of the recurrent nerves.

The muscles supplied by the latter nerve may be grouped as abductors and adductors. The crico-arytenoideus posticus on each side is the only muscle concerned in abductor paralysis; therefore, all of the others, that is the crico-arytenoidei-laterales and the arytenoideus are adductors; the thyro-arytenoidei are also

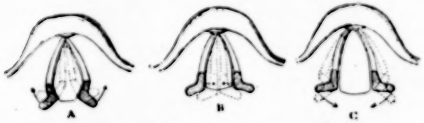
adductors, but their main function is to make the cords tense.[1]

St. Clair Thompson's diagrammatic illustrations (*A*, *B* and *C*, Fig. 1), aid greatly in a clear conception of the function of the various muscles. In the typical normal larynx the edges of the vocal cords are brought together in a straight line in phonation; on quiet inspiration they are separated at the widest part about 7 or 8 mm.; on deep inspiration they are separated about 10 or 11 mm. In the cadaveric position they remain separated about 3 or 4 mm.

Exclusive of ankylosis, if the vocal cord cannot be drawn to the mesial line, there is paralysis of its adductor muscles. If a cord cannot be drawn away from the mesial line, there is paralysis of the abductors; if it cannot be drawn farther from the mesial line than the position of quiet inspiration, there is paresis, or partial paralysis, of the abductors; but if it can be drawn to the position of deep respiration, the abductors cannot be involved.

If a cord on respiration or attempted phonation remains in cadaveric position (about 2 mm. from the mesial line), then both its abductor and adductor muscles are paralyzed.

According to Semon's law, in organic paralysis of the laryngeal muscles, the abductors are always first involved; therefore, if the cord cannot be drawn outward beyond the position of quiet inspiration, we may conclude that it is the beginning of what will before long develop into complete paralysis.



Action of intrinsic muscles of the larynx. *A*. The arrows indicate the direction of action of the crico-arytenoideus lateralis, and the dotted lines show the positions to which its contraction brings the cords and vocal processes. *B*. The arrows indicate the direction of action of the interarytenoideus muscle, and the dotted lines show the positions of the cords and vocal processes on its contraction. *C*. The arrows indicate the direction in which the crico-arytenoideus posticus muscle contracts, bringing the cords and vocal processes into the positions indicated by the dotted lines.

PARALYSIS OF THE CRICOTHYROID MUSCLE

As this is the rarest of all forms of laryngeal paralysis and the only one in the domain of the superior laryngeal nerve, it will be considered first.

Causes, Etc.—This paralysis is associated with paralysis of sensation and may be caused by operations, by pressure on the nerve, or much more commonly by diphtheria. When this muscle fails to act the thyroid cannot be fixed on the cricoid cartilage and therefore, for want of a point of resistance, the vocal cord cannot be put on a stretch, and its edge will remain flabby, with a wavy outline, and will be blown upward in expiration and sucked downward in inspiration. In unilateral paralysis the cord appears shortened and smaller than normal, and it may disappear under the ventricular band in respiration. Paralysis of both cords gives a wavy outline of the glottis. The finger placed over the cricothyroid muscle may detect the feebleness of its action. The voice is feeble and cracked and tires easily.

Treatment.—As anesthesia of the larynx is likely to be present in these cases, care must be exercised to prevent food from entering the trachea. This can usually be avoided by feeding soft solids with the head well below the level of the body; or by feeding only fluids passed through a stomach-tube. If the tube is employed one should make sure that it has not been passed into the larynx, by causing the patient to phonate, before any fluid is introduced. Faradization has been recommended, but carefully watched large and increasing doses of some preparation of nux vomica or its derivatives will be found most beneficial. The tincture of nux vomica appears to me to give the most satisfaction.

PARALYSIS OF THE ABDUCTORS OF THE VOCAL CORDS (THE CRICO-ARYTENOIDEUS POSTICUS)

This being the only abductor of the cords, memory will be aided by considering it at this time. The affection just considered is probably the rarest, but this is the commonest of organic paralyses of the cords. It is caused by (1) bulbar lesions; (2) lesions of the vagus or recurrent laryngeal nerve; (3) peripheral neuritis and (4) inflammation of the muscles.

1. Bulbar Lesions.—As a cause these lesions are infrequent, but may occur from thrombus, embolus, or local aneurysm; from syphilis, tumors or abscess; from diphtheria, general paralysis, locomotor ataxia (bulbar) and very rarely from some other cerebral lesions.

2. Vagus or Recurrent Lesions.—These lesions are the usual causes of this affection and the most common of these are pressure from aneurysm of the arch of the aorta, or pressure as in goiter; also mediastinal tumors, aneurysms of some of the aortic branches, and enlarged bronchial lymph-nodes; cancer of the esophagus and operative accidents to the vagus or recurrent nerve. Conditions said to have caused it are pleural thickening at the apex, especially of the right lung, or large pleuritic effusions and, possibly, growths at the base of the brain involving the vagus close to its exit from the skull; aneurysm of the internal carotid, or abscess in the neck, and possibly pericarditis or dilatation of the left auricle involving the left recurrent nerve.

3. Peripheral Neuritis.—This may result from various mineral or vegetable poisons of which lead is far the most frequent, and very rarely from the toxins of certain infectious diseases as diphtheria, typhoid, influenza and rheumatic fever. Among the exceptional causes are typhus, pneumonia, gonorrhea and erysipelas.

4. Inflammation and Infiltration of the Muscle.—This may occasionally cause this trouble in cancer and exceptionally in injuries from foreign bodies and in syphilis or tuberculosis.

Symptoms and Signs.—Usually there is alteration of the voice and more or less dyspnea; the dyspnea becoming serious if both muscles are involved; sometimes, however, the patient notices no laryngeal symptoms.

UNILATERAL ABDUCTOR PARALYSIS (CRICO-ARYTENOIDEUS POSTICUS)

When partial, this condition merely prevents complete abduction of the cord, but when it is complete, the unopposed action of the adductors draws the cord to the mesial line and the tensors keep its edge straight so that the voice may not be affected and, if it comes on slowly, the patient may not notice any dyspnea. If there is dyspnea some cause should be sought below the glottis. In advanced cases, except those purely myo-

1. It is generally accepted that the thyro-arytenoidei act mainly as tensors of the cords, but the function of the thyro-arytenoidei externi is very uncertain. That these would compress the ventricle appears certain, but their action on the cords, if any, is unknown. It appears to me that these muscles have an important function as adductors of the ventricular bands in closing the larynx against the entrance of foreign bodies; and occasionally in supplementing the action of the true cords.

V. 191

pathic, the adductors also become involved and then the cord remains in the cadaveric position if the paralysis is complete, or, when there is only paresis, in the position of quiet inspiration, in which case the cord appears relaxed, its edge concave and its surface narrow.

The condition may not be recognized in quiet respiration; but in deep inspiration the cord does not abduct as far as its fellow. If the adductors are also involved, as is usual, it is not drawn to the middle line in phonation, while the sound cord is likely to pass beyond the middle line; thus the voice may remain good although it is usually cracked. The arytenoid either remains motionless or is pushed over by the movement of the sound side. The voice is likely to improve with time because of compensatory action of the sound side. Grünwald says that frequently there is twitching of the affected arytenoid cartilage due to contraction of the interarytenoid muscle which is innervated partly from the opposite side. If this twitching is present it at once excludes ankylosis.

BILATERAL ABDUCTOR PARALYSIS (CRICO-ARYTENOIDEI POSTICI)

This is not so common as the affection of one side only. When partial, as it usually is in the beginning, the cords are separated a little way on deep inspiration so that respiration may not be greatly interfered with and phonation may remain normal; but when complete, unless the adductors are also paralyzed, the cords remain in apposition and dyspnea becomes so urgent as to demand intubation or tracheotomy. In this condition the cords appear to be sucked closer together on inspiration, but the voice may be fairly good. When the adductors are also completely paralyzed the cords remain in the cadaveric position and respiration is not so much impeded. All patients with bilateral abductor paralysis are subject to sudden and dangerous attacks of dyspnea.

Diagnosis.—This can generally be easily made on inspection by observing the foregoing points, but there is sometimes much difficulty in distinguishing this condition from ankylosis. Differential diagnosis between these two rests on the following points: A history of inflammation with swelling and induration without evidence of nervous origin is usual in ankylosis; not so in paralysis. The arytenoid cartilage is not pushed over by its fellow in ankylosis on phonation as it often is in paralysis. Any movement of the cord is incomplete, or the cord is quite immobile even with the probe and it may be in an unusual position in ankylosis. In paralysis there may be twitching of the arytenoid inward by the interarytenoideus muscle; the cord, early in the affection, is likely to be in the mesial line, but, later, in the cadaveric position and it is movable with the probe.

Treatment.—If the history points to an origin in syphilis, lead-poisoning or tuberculosis, appropriate general treatment should be adopted. In other cases strychnin in gradually increasing doses to the physiologic limit should be tried thoroughly. The spasmodic dyspnea may sometimes be relieved by the administration of bromids or antipyrin or by inhalation of amyl nitrite.

For the relief of the dyspnea resulting from inability of the abductors to open the glottis it has been proposed to excise the cords, but the results are not good. Intubation may give temporary relief, but unless the affection soon yields to treatment tracheotomy must be done. A tube with cap and valve will allow the patient to breathe and also to use his voice. If due to goiter, removal of the isthmus will usually give relief, but if the gland is large a considerable portion of it should be removed.

ORGANIC ADDUCTOR PARALYSIS

This affection of the vocal cords is a sequel of and due to the same causes and requires the same general treatment as abductor paralysis. The appearances caused by this affection will be the same as those in functional paralysis, which will be described below.

PARALYSIS OF THE ADDUCTORS OF THE VOCAL CORDS

This includes paralysis of the crico-arytenoidei laterales, the interarytenoideus and the thyro-arytenoidei.

Unilateral Paralysis of the Crico-Arytenoideus Lateralis.—This is extremely rare and is due to local causes. If it were only partial the cord would approximate the mesial line more or less in phonation and might remain as in quiet respiration, moving slightly in respiration and phonation. If complete, the cord would be drawn outward to the limit of the abductor muscle, giving it a concave border as in deep inspiration. This paralysis is sometimes produced by acute catarrhal laryngitis. I have seen at least three cases of this kind; but the affection has also been attributed to lead-poisoning, syphilis, typhoid and small-pox.

Diagnosis and Treatment.—Having excluded ankylosis of the arytenoid the position of the cord at the side of the larynx in attempted phonation would establish the diagnosis. Treatment should be directed to the cause and strychnin may be very useful.

Bilateral Adductor Paralysis of the Crico-Arytenoidei Laterales.—When of organic origin this results from inability of the recurrent laryngeal nerve, or the vagus from which it is given off, to transmit motor impulses to these muscles. It is due to the same causes as abductor paralysis, with which it occurs in the later part of all distinct manifestations. The proper treatment has already been indicated. The diagnosis rests on the history and the same appearances as shown in functional affections.

FUNCTIONAL ADDUCTOR PARALYSIS

This affection is known also as functional aphonia and nervous or hysterical aphonia. Nearly all cases of this affection are hysterical, but myopathic causes may sometimes be found. They result from acute catarrhal inflammation or straining of the voice and cases have been attributed, exceptionally, to lead, arsenic, phosphorus or copper poisoning and very rarely to syphilis, tuberculosis, typhoid, etc.

The affection comes on suddenly and may be brought about by any of the many exciting causes of hysterical attacks. Often there is a history of previous attacks. The speech is reduced to a whisper and in very aggravated cases may even be lost. There will be no resultant cough or dysphagia and no pain or evidence of constitutional disease except common symptoms of anemia, neurasthenia and debility which are often present. In cases resulting from debility the symptoms come on more gradually, the voice possibly being weak for several days before it is lost.

Diagnosis.—Usually the cords are found in the position of quiet respiration, but they will be still further drawn apart on deep inspiration. On attempted phonation they are not brought much nearer together than the cadaveric position, although they may be fully approximated for an instant to separate again before any sound is emitted.

Often the laryngoscopic examination alone is sufficient to cause approximation of the cords when the patient is asked to phonate; but if the patient does not

respond to the request to say *A* she should be induced to laugh or cough. Usually the application of a slightly astringent spray as the patient attempts to phonate will bring the cords together at once and the cure will have begun. Whenever in such a case the cords are seen to approximate closely, whatever means may have been employed, the diagnosis of functional paralysis is established.

Treatment.—Measures appropriate to the cause should be instituted and generally a few applications of a mild astringent as zinc sulphate, 2 grains to the ounce, will aid in the cure. Nothing in my experience, however, has equaled strychnin in regular and increasing doses until the result is accomplished. I usually begin with 1/30 grain three times a day and gradually increase it until sometimes I give as much as 1/8 grain three times a day. The patient should be informed of the probable physiologic effects and directed to reduce the dose as soon as muscular spasms occur. The remedy should be continued in doses just below those causing toxic symptoms until recovery is complete. Thompson advises encouraging the use of the voice in reading aloud, talking and singing and especially advises avoidance of the use of electricity, as the latter is so uncomfortable that the patient is liable not to return for any treatment in case of a relapse.

PARALYSIS OF THE INTERARYTENOIDEUS MUSCLE

This may be associated with paralysis of the thyro-arytenoidei or may occur alone. It is usually due to acute catarrhal inflammation, but paresis of this muscle may be hysterical. It causes a triangular chink back of the vocal processes in phonation. Treatment should be directed to the causative laryngitis or nervous debility.

BILATERAL PARALYSIS OF THE THYRO-ARYTENOIDEI

This causes gaping of the cords from 2 to 4 mm. in front of the vocal processes during phonation and makes the voice very weak and hoarse. It should not be forgotten that in some normal persons there is gaping of the vocal cords in phonation of from 1 to 1.5 mm. In my experience this affection has practically always resulted from acute laryngitis; I have seen, however, one chronic and persistent case caused by an attack of measles several years previously. The affection is sometimes very persistent and in such cases proper voice exercises and correct methods of speaking and singing should be secured.

Persevering use of the faradic current is also recommended and strychnin should be tried thoroughly.

104 South Michigan Avenue.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. MAYER, BECK, DENNIS AND INGALS

DR. THOMAS HUBBARD, Toledo, Ohio: Intratracheal injection is useful as an aid to direct treatment of chronic laryngitis. It is usually essential to reach the whole laryngotracheobronchial tract. The use of the syringe has not been generally satisfactory because the instruments on the market are not practical. I use a cannula with a very short curve and make no attempt to introduce it into or beyond the glottis; in fact, I do not even touch the epiglottis. Under the guidance of the throat-mirror it is easy to drop medicated solutions through the glottis without causing any irritation and thereby reach the lower part of the tract and treat it without irritation of the larynx. The larynx is thus thoroughly cleansed from below upward. I have simplified the operation somewhat in certain cases in which it was desirable that the operator should hold or depress the tongue. For these the mirror is attached to the cannula. The syringe should have a capacity of about 16 gm. (4 dr.). The ordinary glass syringe will do. You can pack with asbestos to secure easy, smooth action. This kind of syringe can readily be sterilized by boiling.

A pure mineral oil is the best menstruum in the treatment of these conditions. The oil should be tested with iodin crystals, which produce a permanent cherry-red color. In impure oil the color fades in a few days. Menthol and guaiacol, 3 per cent., in Russian petroleum oil, or weak solutions of iodin can be used in quantities varying from 4 gm. to 8 or even 12 gm., depending on indications. Subacute and chronic laryngitis is very often complicated and occasionally caused by bronchitis or tracheitis. Treatment should be directed toward removal of such complication or cause, and I consider intratracheal injections highly efficient if proper attention is given to indications and technical detail.

DR. LEROY S. PETERS, Silver City, N. M.: In the New Mexico Cottage Sanatorium we treat many cases of tuberculosis of the larynx. Fifteen years ago the cases presenting laryngeal complications were considered fatal. I think that this was due to a lack of early diagnosis. In laryngeal, as in other cases, an early diagnosis enables us to get better results in our treatment.

Dr. Dennis' point with reference to the paleness of the larynx as a diagnostic sign is well taken. Seldom do we see in our routine examinations a pale larynx, but, on the other hand, they are highly red and inflamed. The tuberculin test may be valuable in establishing an early diagnosis by causing a local reaction, although, as a rule, in giving tuberculin therapeutically when a slight reaction arises we see very little change, if any, in the larynx.

In making a differential diagnosis between tuberculosis of the larynx and other diseases, syphilis causes us more difficulty than any other disease. Let me cite a case that I had under observation. The patient was in good physical condition, was of normal weight, but was running a temperature of 103 F. He had a bilateral apical lesion with an involvement of the larynx from the base to the upper part of the pharynx. I made a tentative diagnosis of syphilis. Two doses of salvarsan were administered, however, in one week and I was unable to influence the course of the disease in the larynx. A Wassermann test proved negative. After the salvarsan injections the patient began to lose strength and in two weeks was dead. That man probably had a rapid type of laryngeal tuberculosis, although the diagnosis proved difficult.

I commend the attitude taken by Dr. Dennis as to the treatment of laryngeal tuberculosis, against the stand laid down by so many that these cases are as well let alone. The results obtained in sanatoriums may be partly due to the rigid régime prescribed for the patient. However, the local applications that are made to the throat twice daily in our routine work are, we believe, responsible for the large percentage of cures we have in this class of cases. We use tuberculin therapeutically believing that it is as good in laryngeal tuberculosis as in the pulmonary form.

Dr. Dennis has spoken of the treatment of the cough. I believe that we should not give sedatives, especially codein or any of the morphin derivatives. The use of orthoform as proposed gives no results except those that are palliative. Cocain may be used to alleviate the marked pain which follows ulceration. In the treatment of all of these cases we confine ourselves to the use of liquor formaldehyd starting with two drops to the ounce and increasing to ten drops as tolerance is established. This solution is applied in the non-ulcerative cases with a curved tipped laryngeal spray and, in the ulcerative cases, on a pledget of cotton with laryngeal forceps. We have confined the surgery of the larynx so far only to hopeless cases and then only as a palliative measure. As a hurry-up measure, surgery is indicated and may save much valuable time.

DR. MICHAEL P. SCHUSTER, El Paso, Tex.: The supervision of these tuberculous cases by the laryngologist is important. There are hundreds of sanatoriums that have not sufficiently controlled the laryngeal conditions in tuberculosis. Almost every tuberculous patient has something the matter with the

V. 191

larynx. In the beginning of this disease, in many cases, the laryngeal treatment is all that is needed. The cough is controlled and the patient can live more comfortably; often it is the tuberculosis of the larynx that kills and not the tuberculosis of the lungs.

In the differential diagnosis between tuberculosis of the larynx and syphilis of the larynx, there is one point which has always been a help to me and which has always seemed to point to a correct diagnosis, namely, that in all cases of tuberculous laryngitis there was extreme sensitiveness to touch, while in syphilitic laryngitis this was not the case; sometimes I have even thought that the parts were almost anesthetic.

I have not found salvarsan to be of any help in the treatment of syphilitic laryngitis. The cough should be relieved. It has been estimated that the blast of air passing through the larynx in an attack of coughing goes at the rate of two miles a minute and this, of course, must cause much friction in the larynx. In many patients training will accomplish much. Sometimes if they cough but once in the morning they will be comfortable the remainder of the twenty-four hours. Should these cases be treated locally or not? There are two groups of cases, those which are sensitive and will react and those which will not react; those that react should be left alone so far as any local treatment is concerned.

DR. HOWARD F. PYFER, Norristown, Pa.: What is the value of high altitudes, dry air and continuously sunshiny days in these tuberculous conditions of the larynx? Are we justified in sending our patients to distant states, New Mexico, Texas or Colorado, far from home and its comforts and care? Are the advantages commensurate with the annoyances and irritations?

DR. CULLEN F. WELTY, San Francisco: I wish to emphasize what has been said about pain. This pain is best relieved by injections of alcohol, followed by the proper intralaryngeal operative procedure. Some of my colleagues in San Francisco have used the alcohol injections in a series of cases and comment on it very favorably. The most important thing, however, in laryngeal tuberculosis, is to keep the patients eating all the time.

We should operate for a cure (a) in all cases of primary tuberculosis of the larynx, unqualifiedly; (b) in cases of laryngeal tuberculosis, in which the patients are not losing weight, and do not have a temperature of more than 100 F., that is, in cases in which the lung tuberculosis is more or less quiesent. In some few instances, this operative interference seems to light up a new infection which is, of course, serious. We should operate for the relief of distressing symptoms such as pain, etc.; many patients can be made quite comfortable for at least the remainder of their lives by the proper operative interference.

DR. J. HOLINGER, Chicago: Chronic laryngitis, described by Dr. Mayer, is a difficult proposition. The erosions which extend over the pharynx and larynx often baffle all attempts at a cure. Several cases which I have followed for many months turned out to be carcinoma, yet in the beginning there were no indications of malignancy. One case of gumma of the larynx was of personal interest to me. A man, aged 40, had not been able to sleep for many nights on account of difficulty in breathing. He came to me in an exhausted condition. His voice was almost inaudible. An examination being impossible, I intubated, after which he slept for fourteen hours. When he awakened he coughed out the tube. It was replaced and he slept for another ten hours. He was then examined and a tumor was found on the arytenoid surface which almost completely filled the larynx. The mucous membrane covering it was normal. The diagnosis of gumma was corroborated by Dr. Louis E. Schmidt from a scar on the penis and swollen glands in all characteristic places. Specific treatment was instituted, and at once I had difficulties to contend with. The patient was utterly unscrupulous, disregarded all advice and recognized no law and no opinion but his own. From the beginning he staunchly denied the syphilitic nature of the disease and only reluctantly took antisyphilitic treatment. Against my advice he continued to chew tobacco and smoke, and as a consequence I could not push the mercury. After one week his teeth became loose. He was intubated regularly for several weeks. Repeatedly when the tube came out the interns tried to reintroduce it; other colleagues tried also. Soon the interns refused to treat the patient, because during one of these intubations he gave one of them a blow in the stomach. One evening I could not reach his bedside until an hour had elapsed after the tube came out. I found him unconscious, with shallow, irregular respiration. Owing to the great distances in Chicago, and the possible irregularity of the telephone, I could not carry the responsibility any longer, and insisted on tracheotomy, but he refused. After several days, however, when the alternative was put to him, either to leave the hospital and engage another physician, or to consent to the operation, he reluctantly submitted. Afterward he was sulky because he could not talk at all. In the spring he went to Arkansas, whence he returned much improved in his general condition and wanted the tracheotomy tube removed. By laryngofissure, I removed the gumma submucously. The result was perfect. One could see only a small scar in the anterior wall of the larynx. After the operation, I found that, owing to the vocal cords being totally paralyzed he could not breathe without the tracheotomy tube. I proposed either to wait for a possible recovery of the motility of the cords, or to excise the cords. He then disappeared and I did not hear from him again until five months later, when he came into my office, pulled out a pistol and committed suicide. He was a professional slugger for the plumbers' union.

DR. BURT R. SHURLY, Detroit: It seems to me that the point made that we should have a laryngologist in every hospital devoted to tuberculosis (and we seldom hear of an attending laryngologist) is a timely one. There are a few sanatoriums in this country in which it is recognized that the use of the knowledge of a laryngologist is worth while. It seems to me that this study of the larynx is essential. The older laryngologists were laryngologists in every sense of the word. There is a danger that we may wander from this interesting field. I have seen a large number of cases of laryngeal tuberculosis and have sent these patients for climatic treatment to those who were particularly interested in this disease. In looking up the statistics of patients who have been under observation for seventeen years I find that 96 per cent. of those with incipient cases are alive and well to-day. I have never reported that record because I do not believe that such a report is valuable until patients remain alive and well for periods of at least from five to ten years; for we know what an arrested case is, and we know what may occur from day to day and from year to year. The laryngologist has a definite procedure in the treatment of this particular class of cases.

I do not believe that the internist should tell us how to treat syphilitic laryngitis. He should not tell us when to take the tonsils out. These questions belong to our particular domain. Our knowledge should be so definitely perfected that the internists would come to us and ask us how to treat the larynx.

DR. ELMER L. KENYON, Chicago: In the routine examination of several hundred patients at the Edward Sanatorium for tuberculosis, I have been struck with the frequency of the occurrence of enlargement of the soft tissues in the region of the posterior commissure of the larynx and of the arytenoepiglottic folds. I formerly considered such swellings of grave tuberculous importance. And so they may be. Apparently, however, the pathology in these cases is not always the same. In these relatively early cases of pulmonary tuberculosis probably 50 per cent. show swelling of this character and yet the larynx in a large percentage of these cases gives no trouble and the patient recovers. The local irritation arising from outdoor living in cold weather may account in part for the condition, and I also think it possible that the secretions from the lungs sticking in the laryngeal region may produce irritation of the mucous membrane and cause laryngeal efforts at expulsion, resulting in local edema. At any rate, many of

these enlargements in the early stages of pulmonary tuberculosis seem not to possess grave importance.

Regarding the differential diagnosis between tuberculosis and carcinoma of the larynx the following case is worth considering. A young, vigorous man, aged 30, with a family history clear of both tuberculosis and carcinoma developed a chronic enlargement of one vocal cord. The chest was negative. The von Pirquet test gave a positive reaction. In consultation with Dr. Casselberry his physician employed the tuberculin test by injection; the temperature rose to 102 F. and a local hyperemia appeared in the larynx. A working diagnosis of primary laryngeal tuberculosis was made and local applications of liquor formaldehyd instituted, with similar treatment later in Asheville. The ultimate outcome, however, was the total extirpation of the larynx for epithelioma after four years.

Here was a young man with a slowly developing lesion of the larynx, giving positive tuberculin reaction, both general and local, and yet whose disease turned out ultimately to be carcinoma. Was the primary condition epitheliomatous, or tuberculous, or neither?

DR. LEROY S. PETERS, Silver City, N. Mex.: I wish to correct an erroneous idea of altitude as entertained by one of the speakers. The elevations in New Mexico range from 3,000 to 9,000 feet, differing little from those of Colorado. With such elevations one could hardly call New Mexico a low altitude state. Silver City has an altitude of 6,000 feet. I think the consensus of opinion has been that patients with laryngeal tuberculosis do not do well in high altitudes. However, Dr. Shurly's statistics show that we get very good results in cases of laryngeal tuberculosis treated at an altitude of 6,000 feet, and the results obtained by him in referring his patients to us should answer the question relative to altitude treatment of laryngeal tuberculosis.

DR. ROBERT LEVY, Denver: I wish to emphasize the importance of the local treatment of laryngeal tuberculosis and especially the relief of pain by the injection of alcohol into the superior laryngeal nerve. That of itself is not curative, but it enables the patient to take nourishment and also permits of more satisfactory treatment. One can get at the larynx and at the local lesion with greater ease and accuracy.

I also wish to lay stress on the treatment of laryngeal tuberculosis both in the infiltrative and the ulcerative variety by the galvanocautery. I do not know what limit one should place on the use of the galvanocautery, but it can be applied to all so-called surgical cases. The galvanocautery is also applicable to those cases which are manifestly progressing, cases in which there is edema and in which small yellow or gray spots of tubercles appear under the mucous membrane. These are the cases I have always considered unsuited for local surgery, but even they lend themselves to treatment by the galvanocautery. In many very acute cases I have taken it on myself to stick the galvanocautery into the mucous membrane and in several instances I have obtained gratifying results. I wish to urge those who have not tried the use of the galvanocautery to do so, especially by deep puncture in the infiltrative variety. I urge you to try it in those cases which give you the greatest trouble. The puncture is not only of advantage at the immediate site of the puncture, but it has a beneficial effect on the surrounding tissues as well; they will undergo a fibrosis, then a contraction and eventually, in a number of instances, a reduction in the size of the swelling, a passing of the infiltration and a restoration of function will be seen.

DR. EMIL MAYER, New York: If singers or public speakers come to you, it is all-important that you give them relief promptly as it often affects their means of livelihood. I have not used intratracheal injections in these laryngeal conditions. I have found them of advantage in tracheal, but not of so much advantage in laryngeal conditions that cannot be helped otherwise. In the syphilitic cases mentioned by Dr. Beck I have never found it necessary to perform laryngostomy. These cases of chronic stenosis can be cured by intubation and dilatation. The use of Killian's suspension laryngoscope is of great advantage, and with it we can often avoid the tedious and distressing methods usually employed in treating these patients.

The pale mucous membrane is usually looked on as suspicious and gives us something which is an aid in the diagnosis. There is one condition that has not been mentioned here that we should be on our guard against. It is best shown in a brief report of a case. A patient was brought to me a short time ago by a physician who stated that the patient had some deposit on his tonsil. The patient had been coughing for some time, but he had no pain, swelling or huskiness of voice. When I examined him I found an extensive destruction of the epiglottis. What was left was rough and raw and the arytenoids were infiltrated. The patient had been treated for two years for tuberculosis, but never before had he had his larynx examined. The statement has been made that in laryngeal tuberculosis pain is a symptom, but this was not true in this case. I diagnosed lupus of the larynx. I had had a patient with lupus of the larynx eighteen years before who succumbed to tuberculosis. It is surprising to see the tremendous amount of destruction which can go on. The real process in these cases I believe to be the lupus of the larynx.

I would say by all means operate on these patients even though you know that the ultimate end is near. If you can relieve the patient by euthanasia you will be doing him a good turn. With the Killian suspension laryngoscope you can take off the whole epiglottis. Patients will do well without the epiglottis. They do well even when the galvanocautery or some other instrument is used. The results are certainly surprising.

The statement has been made that every sanatorium in the country should have a laryngologist connected with it. Many of us will not refer a patient to any sanatorium that does not have a laryngologist see the patient from time to time.

DR. J. C. BECK, Chicago: The cases of laryngectomy are those which Dr. Mayer referred to and which should be treated by a competent laryngologist. It should be remembered that these patients may die from lack of air, and the results of laryngectomy show that they are relieved and are very satisfactory; the operation is easily carried out.

DR. MICHAEL P. SCHUSTER, El Paso, Tex.: I have used salvarsan and am willing to testify to the value of this drug. One point that has not been brought out or mentioned is that sometimes the injection of the superior laryngeal nerve is unsatisfactory. This is so not only in tuberculous cases, but also in malignant cases following operation on the epiglottis. Injections of alcohol will not give relief in every case; whereas resection of the nerve is followed by absolute relief.

I removed a larynx five or six days ago in a case in which a positive Wassermann reaction was made. The patient was treated extensively with salvarsan and with other antiluetics. The condition proved to be an epithelioma.

DR. F. L. DENNIS, Colorado Springs, Colo.: Bandelier and Roepke speak quite favorably of the influence of tuberculin though they admit that the choice of case must be made with the most careful consideration of the state of the lungs and the general condition. Further they state that tuberculin treatment of laryngeal tuberculosis usually takes a long time, since there is generally severe disease in the lungs also which draws, by virtue of its larger area, most or all the tuberculin to itself, and only after the lungs are gradually saturated is the tuberculin free to act on the larynx. So far as my experience goes I have never seen any direct results from tuberculin in these cases.

I am convinced that narcotics are bad in cases of tuberculosis. On the other hand, cough is bad in laryngeal tuberculosis. By every means in our power we should remove the irritation which causes the cough.

Food Injuries.—The food injuries of Czerny, his fat injuries, his sugar injuries and his protein injuries are the old familiar fat, sugar and protein dyspepsias of Rotch and Holt, but writ large and in capital letters.—Eric Pritchard, in *Clin. Journ.*

V. 61
1913

THE RESULTS OF TONSILLECTOMY UNDER LOCAL ANESTHESIA. NEW OPERATION *

BRYAN D. SHEEDY, M.D.
NEW YORK

It seems to me wholly unnecessary to give a long introduction to this subject in speaking before specialists who have been for years following this special work. I know that every man here has removed tonsils time and time again, under local or general anesthesia or with

Fig. 1.—Normal tonsils.

no anesthesia, with what he considered satisfactory results to both himself and his patients.

The length of time that has passed since a given operation is an important consideration in examining throats as to the results found following tonsil enucleation, so I desire to state that in all of the cases reported operation was performed not less than three months before examination and that in no patient under 14 years of age was local anesthesia employed. I have not separated into groups patients in whom the tonsils

Fig. 2.—Right anterior pillar gone; scar tissue on posterior; contraction of posterior pillar; large mass of scar tissue where left anterior pillar should be.

were enucleated under local anesthesia in contradistinction to general anesthesia or no anesthesia.

To introduce the discussion I shall report the results of examination of 100 patients on whom tonsil enucleation was performed by some one of the methods now in vogue, these examinations having been made at my clinic and in my private work during the past eight to ten months. There have been some of the operations performed in almost every public institution in New York City and Brooklyn and hardly a man of prominence in those institutions was omitted from the list of operators, though a fair percentage of the patients were operated on by men with only limited training and experience.

The results of tonsil enucleation, that is the removal of the tonsil with its capsule complete (tonsillectomy), are, I believe, so far as removing diseased tonsils is concerned, very much better than the results obtained from the old tonsillotomy procedure by which only a portion of the gland and capsule was removed. Though we now have many throat deformities and defects following the newer method the constitutional benefits derived from the procedure are, I believe, far greater than and outweigh the drawbacks following the work, as shown by throat deformities with singing and voice defects. There is no doubt in my mind but that many of the physical throat deformities are due to the present clumsy and non-surgical methods employed in the removal of the gland, so if a single case of deformity is prevented by the "eversion operation," as a result of this discussion, I shall feel repaid for digressing from my text for its introduction. To emphasize the points necessary to an understanding of complete local anesthesia, as well as

Fig. 3.—Complete absence of both pillars on right side with soft palate drawn up; absence of left posterior pillar with marked contraction of left anterior pillar; large masses of scar tissue where pillars were located; space between soft palate and pharyngeal wall very much limited.

the removal of the tonsil by the "eversion method," your minds must be clear on the anatomic make-up of the gland and its surroundings. Therefore, I shall briefly outline to you the anatomy of the parts, quoting liberally from the subject as presented before this Section by one of our esteemed confrères on a former occasion.

ANATOMY OF THE STRUCTURES

Whether the tonsil should be called a gland or the gland a tonsil, or whether it should be designated simply as lymphoid tissue or given some other name, will not concern us in this discussion. I shall use the terms interchangeably and consider the tonsil a mass of glandular tissue located between the anterior and posterior pillars, but quite independent of them when in a normal condition. The outer wall of the bed on which the tonsil rests is, as you know, made up of the superior constrictor of the pharynx over which there is a thin layer of connective tissue.

The gland, we are told, first appears at the fourth month of intrauterine life and reaches its fullest development at from 5 to 7 years of age, at which time it begins

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

to atrophy and, if it has not been diseased, should have wholly disappeared by the time the child is 15 years old. This gland is only a part of the lymphoid tissue derived from the hypoblast and it is important that we should keep this matter in mind, as too many physicians are inclined to consider disease of the faucial tonsils a local one without bearing in mind that the mass in the nasopharynx, which is called adenoids, or that found at the base of the tongue, described as the lingual tonsil, or the glandular tissue in the crypts of Morgagni, or the peptic glands of the stomach, or the lymphatic structures of the duodenum, or the mass going to form Peyer's patches, or that so often filling the appendix, are lymphoid structures and all derived from the hypoblast, so that when there is a tendency to hypertrophy and abnormality of one portion of the lymph structure, we should keep in mind the possibility of abnormality of all parts in which hypoblastic formations are found.

The tonsil, we are told, is wholly covered by the plica tonsillaris before birth, but shortly after birth the membrane disappears from that part of the gland afterward exposed in the oropharynx. The remainder of the gland is surrounded by a dense layer of connective tissue, generally described as the capsule. Therefore, the capsule extends around all of the gland, except that portion

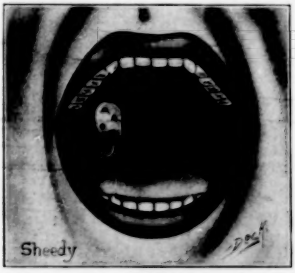


Fig. 4.—Tonsil everted through hypertrophy of its structure.

opening into the throat. We find on the inner side of this capsule bands of connective tissue extending from one side of the capsule to the other, and, suspended on this connective tissue, we find the glandular structures. Leading into the tonsil from the space over which there is no capsule are several openings, according to the size of the gland, which we call crypts or lacunae, and we find that the mucous membrane covering the pharynx extends over the pillars and into these pockets and, through its adherence to the surrounding structures, helps to hold the tonsil in position.

When we examine more carefully the tissues surrounding this gland, we find that the anterior pillar is formed by the muscle, which runs from the soft palate to the base of the tongue, the palatoglossus muscle. The action of this muscle constricts the opening between the mouth and the pharynx and is concerned with the position of the soft palate in the act of swallowing and talking. Frequently inflammatory adhesions are found between this muscle and the tonsil. This muscle, when the tonsil is not very large, is rather thick and appears conspicuously, in the act of gagging or when the tongue is brought forward, through the mucous membrane covering it. When the gland has been hypertrophied for a great length of time we find this muscle, or in other words the anterior pillar, very thin with only a few shreds of the former muscular tissue remaining.

Posterior Pillar.—The posterior pillar is also made up of a muscle, which runs from the soft palate above to the pharyngeal wall behind, where a portion is inserted into the aponeurosis; while another, and the principal part, passes forward and is inserted into the thyroid cartilage. This muscle has much to do with properly locating the larynx during the act of singing. The combined action of the pillars on the right and left side of the throat with the pad of Passavant, almost, if not completely, shuts off the mouth from the upper respiratory tract. The membrane of the pharynx extends over the posterior pillar the same as over the anterior pillar, holding the gland in position.

Supratonsillar Fossa.—The tonsil bed between the two pillars and above the superior surface of the tonsil, when in normal position, is generally described as the supratonsillar fossa and is covered by the same mucous membrane that covers the tonsil. In this space we find openings leading into the cellular tissues on which the tonsil rests and in which we have suppuration in cases of peritonsillar abscess. It is important at this point to remember that there are one or two crypts in the top of nearly every tonsil, which open into this space and

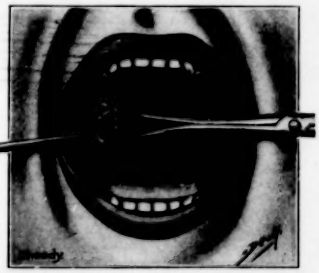


Fig. 5.—Tonsil everted. Snare in position.

which are frequently the cause of infectious material getting into the sinuses leading to the cellular tissue and causing peritonsillar abscess.

Tonsil Bed.—That portion of the tonsillar fossa actually occupied by the gland rests against the superior constrictor muscle of the pharynx, but between the muscle and the capsule is a layer of cellular tissue so that there is a layer of cellular tissue all around outside the capsule and between it and the muscles surrounding the tonsil. This is the tissue in which local anesthetics should be placed when the tonsil is removed under local anesthesia, so that the gland really rests on a mass of cellular tissue infiltrated with the solution used to bring about the anesthesia. As we look at the bed of the tonsil we find three large arteries leading to the gland: (1) the tonsillar artery, a branch of the external maxillary; (2) the ascending pharyngeal, which is a branch of the external carotid, and (3) the descending palatine. There are several smaller branches, but the three enumerated are the ones we should have in mind in dealing with hemorrhage. It is well to remember, too, that in a large hypertrophied tonsil all the blood-vessels are enlarged and, therefore, many of the smaller vessels become very prominent. Without taking up too much time emphasizing the anatomic relationship of

V. 61
1913

the tonsil, I want to impress on you the outline of the pharyngomaxillary space, which lies behind and outside the tonsil and in which there is a large amount of cellular tissue and in which kidney-shaped space the blood-vessels are found and in which we have the accumulated pus in peritonsillar abscess. The shape and location of

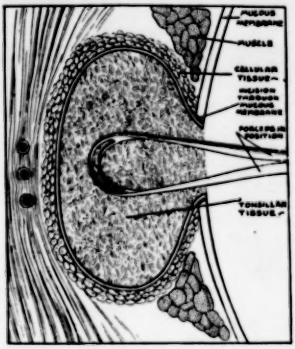


Fig. 6.—Position of forceps before traction is applied on tonsil in eversion operation.

this pharyngomaxillary space indicates why we should open our peritonsillar abscesses at the point midway between the base of the uvula and the last molar tooth, where the space comes nearest the surface. The anatomic relationship between the blood-vessels and this space also explains the serious hemorrhages that we frequently have in peritonsillar abscesses. As the nerves are found with the blood-vessels it is readily appreciated that if this cellular tissue is infiltrated with a watery

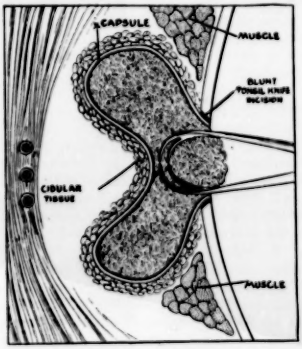


Fig. 7.—Tonsil everting.

solution there will be a minimum amount of pain in the removal of the tonsil.

After this review of the anatomy of the parts we are prepared to discuss the various forms of operation for the removal of the gland that have been in vogue for some time past. Without going into the matter too much in detail, I would say that the aim in all the tonsillectomy operations has been the complete removal of the gland with its capsule and this has been accomplished (1) by taking hold of the gland with a pair of tonsil forceps and pulling the whole gland from its bed, dissecting away with sharp knives or scissors the mucous membrane and cellular tissue; (2) by making an incision through the mucous membrane and then bluntly dissecting with the finger and thereby tearing through the cellular tissue; (3) by extracting the tonsil from its bed, incising the mucous membrane and then passing the snare or tonsillotome outside the tonsil, thus removing the whole tonsil and capsule; or (4) by forcibly dislocating the tonsil with its capsule from its bed on to the inner surface of the lower jaw-bone and then removing with a special instrument designed for the purpose.

The above-mentioned procedures, or some modification of them, have been for some time the customary methods for the removal of the faucial tonsil.

Of the series of 100 cases (ages of patients 14 to 40) examined from three to ten months after operation, in which some one of the above forms of surgical pro-

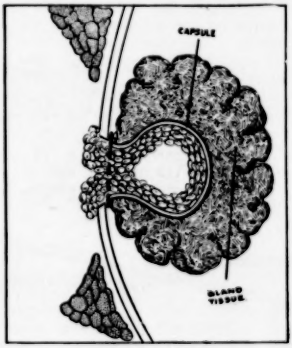


Fig. 8.—Everted tonsil.

cedure was employed, deformed throats were found in more than 80 per cent. of the patients and not more than 20 per cent. had throats approaching the normal. In the 20 per cent. with normal-appearing throats the speaking and singing voice, so far as the patients knew, was normal from date of removal of the gland and they had no trouble whatever following the operation. Of the eighty patients, thirty-four complained of speech defects for two or three weeks, sixteen complained of speech and voice defects three months after operation, four practically lost the singing voice, while twenty-six said that they could speak better and their throats felt better after operation than before. About 5 per cent. of the whole number complained of difficulty in using certain words and had nasal intonation six months after operation.

The deformities following enucleation seem to follow one of three varieties:

1. The pillars on both sides seem to have disappeared, leaving a flattened surface where the tonsil and pillars formerly were and a much-narrowed opening into the nasopharynx.

2. The pillars on both sides seem to have grown together, leaving but one pillar where there were for-

merly two, with the uvula pulled to one side or the other.

3. The anterior pillar has wholly disappeared and a large amount of cicatricial tissue is deposited on the surface of the posterior pillar, altering its shape and function.

In some of these cases the soft palate and uvula were pulled down to a very much lower level than normal, thus accounting for the nasal intonation complained of by some of the patients. In all the patients in whom the singing voice was affected the posterior pillar was very much out of normal through the contraction of cicatricial tissue. That you may get a better idea of the appearance of these throats I have had composite drawings made from a number of patients (Figs. 1 to 4).

From these results I am sure you will agree with me that the last word on tonsil enucleation has not yet been said. I therefore ask you to consider carefully the procedure that has, in my hands, proved to be almost ideal in every way, not only as a means of preventing the unsatisfactory results above outlined but of simplifying the operation for the removal of tonsils.

Fig. 9.—Blunt pointed tonsil knife.

THE OPERATION FOR EVERSION OF THE TONSIL (SHEEDY METHOD)

In adult patients the surface of the tonsil and pharynx is swabbed over with a 10 per cent. solution of cocain, the pharynx is cocainized to prevent gagging, and after cocainization has been accomplished a solution of bisulphate of quinin, from 1 to 1.5 per cent., is injected into the cellular tissue surrounding and lying outside the capsule of the tonsil. This infiltration causes loss of sensation and the operation is done without pain. In children under 14 years of age ether should be employed.

Fig. 10.—Tonsil tenaculum.

Just as the bottom of a bag may be pulled or pushed through its mouth so that its inner surface becomes the outer, so may the capsule of the tonsil. With a tonsil tenaculum inserted as deep as possible into the center of the gland, *avoiding the capsule*, take hold of the tissues extending from one side of the capsule to the other and make traction until the junction of the mucous membrane with the capsule is apparent. Then introduce a small blunt-pointed tonsil-knife (Douglass) under the mucous membrane at the point of junction with the capsule and incise around the tonsil (this incision later on acts as a guide for the wire when the snare is used), and, if the capsule is so contracted that the gland will not invert, make a nick in the upper angle of the capsule comparable to the nick or incision made in the prepuce to permit the escape of the corona glandis in phimosis, and then with a little more traction endeavor to invert the tonsil, as you would invert a uterus by pulling on the cord of a retained placenta. In 90 per cent. of the cases slight traction will invert the tonsil. After it is completely inverted place the snare around the mass as shown on the chart; then slowly tighten the wire, which slips into the slit previously made at the point where the mucous membrane joins the capsule, take from one to three minutes to cut through the tissues, the tonsil will be removed without injury to the pillars, without pain and without hemorrhage.

I have employed this method for the past two years in a very large number of cases and am yet to have my first case in which the pillars have been injured or in which deformity followed the procedure.

There are some exceptions in cases in which the capsule will not evert and from my experience I would place the exceptions in three classes: (1) that of the exceedingly large tonsil, which we frequently see, in which the hypertrophied tissue escaped from the capsule and by traction everted the tonsil, so that we find the tonsil completely everted when the patient applies for treatment; (2) that in which the oropharyngeal surface of the tonsil is so limited by a contraction of the capsule holding within itself a limited mass of cicatricial tissue that it in no way resembles a tonsil; and (3) that in which the tonsil is only slightly hypertrophied, but in which the cicatricial tissue surrounding the capsule has been displaced by fibrous bands due to previous attacks of inflammation and peritonsillar abscesses.

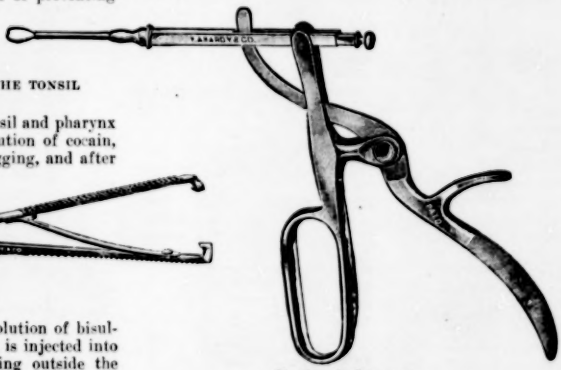


Fig. 11.—Tyding snare.

After two years' experience and after familiarizing myself with the literature on the subject I am convinced that in 90 per cent. of all cases needing operation the proceeding outlined surpasses any measures heretofore known to me for the removal of tonsils.

164 West Seventy-Third Street.

Not Wholly a Medical Problem.—This is but another way of saying that the control and eradication of disease is not wholly a medical problem. It is almost equally a social problem. So long as the general public prefers to submit to the suffering and loss imposed by controllable diseases, medical skill cannot lift the burden.—Corwin and Mayo, in *The Outlook*.

V. 191

TECHNIC OF TONSILLECTOMY

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During the last few years an enormous amount of literature has been written concerning the operation of tonsillectomy, most of which has been occupied with trivial details either of position, instrumentation or order of procedure. If general surgery were to occupy itself with so many minor details for each operation the literature of operative technic would be overwhelming. It seems to me time to simplify somewhat our literature of this subject, and describe principles rather than minute details.

The tonsil is a gland set in each side of the back of the mouth; bounded in front by the palatoglossus muscle or anterior pillar, behind by the palatopharyngeus muscle or posterior pillar, and externally and somewhat above by the superior constrictor muscle, and inferiorly lying against the root of the tongue. It is embraced, except on its free surface, with a fibrous capsule, and this fibrous capsule is attached to the boundaries just described. The operation of tonsillectomy consists in the removal of this gland or tonsil with its capsule intact, and with the least amount of trauma and injury to the adjacent structures, especially the soft palate and the palatine pillars. Any technic which removes the gland in the shortest time, and with the least risk to the patient, both present and subsequent, is a good technic, and that technic is best for the individual operator which he has learned to perform well, and which gives him satisfactory results, whether it be the first, second, or any other of the methods about to be described.

It is essential for any method that the parts be sufficiently anesthetized so that the individual is quiet and free from pain, and the position such as to enable the operator to have at all times a good view of the operative field. Both local and general anesthesia, therefore, come into consideration. In individuals with a fair degree of self-control the injection at many points under the edges of the pillars, at their junction superiorly, and in the tissue around the tonsil of a local anesthetic gives very good anesthesia, and for this purpose, in view of the fact that some unpleasant and even fatal results have followed the use of cocain, it is desirable to use a non-toxic anesthetic. So far as my own experience goes novocain is this anesthetic *par excellence*, since it is non-toxic and can be used in 0.5 per cent. solution. Furthermore, it gives a very good degree of anesthesia, and in subjects in which, for any reason, it is desirable not to use a general anesthetic, it serves very well. Other observers have used with equally satisfactory results the salts of quinin bisulphate or sulphate in 2 per cent. solution, also quinin and urea hydrochlorid. It is claimed for these that the anesthesia is more prolonged and lasts for hours. In young children and in nervous individuals, and even in most adults, general anesthesia is to be preferred, since it allows a thorough and painless operation.

PREPARATION

The preparation should be the same as for any major surgical operation; a previous examination of the urine should be made, the blood-pressure taken, the condition of the bowels and the general health noted, and in women the relation to the menstrual period, as it is desirable not to operate during this time.

If there is any reason to suspect hemorrhage the clotting-time of the blood should be determined beforehand. Young children will not usually require much preliminary preparation. I do not think it desirable to operate during the presence of any acute inflammatory disturbance of the tonsil.

POSITION

Position varies according to the individual preference of the operator. In young children and in adults who are not too big and heavy I am in the habit of using the upright position. Young children sit up in a chair, or in the arms of a nurse; adults sometimes in the chair, sometimes on a table, the head-rest of which is raised sufficiently to give practically this position. The advantage of the position is that with a good source of light the operative field is dryer than in any of the prone positions, and it is easier to see a bleeding point, should there be any. Furthermore, having operated in this way for several years, I am more at home with this position.

Dr. T. R. French of Brooklyn recently invented a table for operating in the upright position, and experiments made by him have not shown that there is any more risk in this position than in the prone one. Equally good work can be done with the head over the end of the table, the light being thrown in from above, or with the head on the side and at the edge of the table, the light being thrown in from the side, and the lower tonsil being operated on first so as not to have the blood from the upper one flow over the lower while it is being removed. Opponents of the upright position claim some risk from blood entering the larynx, but I have not found this to be the case. On the contrary, I am inclined to think that the actual bleeding is rather less in this position, as whatever blood comes, being seen immediately, pressure is at once applied to the point whence it comes. Either direct daylight, preferably from the north, or reflected light is provided, as may be desired.

METHODS OF REMOVAL

The methods of removing the tonsil can be divided into five, under some one of which may be included every modification of technic which so far has been described.

1. *Finger Dissection.*—This is the oldest of all methods, having been described before the Christian era, and having been revived several times since by various writers, one of whom I may claim to be. The method consists in insinuating the tip of the finger between the anterior pillar and the edge of the tonsil capsule, detaching the upper one-third of the tonsil, and then inserting the finger between the tonsil and its attachments to the superior constrictor muscle and adjacent fascia. In this way the entire tonsil can be removed. It is advisable that the initial separation be made slowly and carefully, separating the entire attachment of the anterior pillar both upward and downward, and then with the finger between the superior constrictor muscle and the tonsil capsule the tonsil is readily detached above, and posteriorly where it is attached to the posterior pillar. This dissection should be made slowly and carefully, without haste, and when so done can be accomplished with the minimum amount of trauma. The separation of the capsule from the superior constrictor and its fascia presents no difficulties. In a few moments the tonsil is felt to be separated from its attachment anteriorly, above and posteriorly. It remains attached at its base only. It is then grasped with any type of grasping forceps, drawn well out into the center of the mouth, and the snare placed over the part still remaining undetached at the base of the tongue. It is possible to enucleate completely with the finger even at the base, and some operators so do, but it is preferable to complete the final detachment with a

snare. I do not know that the snare has any advantage over a tonsillotome, except that it tends to follow the natural contour of the tonsil better. So far as the question of hemorrhage is concerned, I do not know that there is any material difference. The instruments required for this technic are a mouth gag of good type (I prefer a Whitehead), some form of separating instrument in case the finger is not equal to making the preliminary separation, which is sometimes the case, a pair of grasping forceps, and a snare. A fairly long nail is desirable. This is covered with a rubber glove or finger-cot, which in no way hampers the procedure, so that the finger is as aseptic as any other method. Finger dissection is not suitable for operation under local anesthesia, as the pressure from the finger causes pain beyond the anesthetized area.

2. *Dissecting with Knife or other Dissector; Patient in the Upright Position.*—While this method is susceptible of many slight variations it consists in nothing more than the grasping of the tonsil with some type of tenaculum or forcep, putting it on the stretch, cutting the edge of the capsule where it is attached to the folds of the anterior and posterior pillar, dissecting it from these attachments while still on the stretch with knife scissors or dull dissector, and then detaching it with the snare or some cutting instrument or completing dissection with the knife or scissors.[1] After the tonsil is separated from its pillar attachments, if strong traction is made on it, it tends almost to evert itself. A snare or detaching instrument then placed over it will usually remove it with capsule intact, the snare almost invariably making a clean dissection. This is a nearly ideal method and is applicable in every case no matter what the type of tonsil.

3. *Dissection with Head Over End of Table.*—The operation is done with the head over the end of the table; the light is thrown directly in on the tonsil, which is grasped with some type of forceps and put on the stretch. The separation of the edge of the capsule is made with some form of knife, a simple long-handled, sharp scalpel answering as well as anything else, and the dissection is completed with knife, scissors or some sort of dull dissector, or with strong traction on the tonsil the snare alone readily finishes the dissection. General anesthesia is almost essential for this and the two following methods.

4. *Dissection with Head on Side.*—The head is turned on the side, but the details of the actual removal are essentially the same as in the preceding, the lower tonsil being removed first. The minutiae of detail by these methods is incident to the individual operator, and depends entirely on his personal preference. It is desirable, though by no means necessary, to have two snares, when one may be left in position while the second tonsil is being dissected; the first tonsil is then detached, and then the second.

5. *The Method of Sluder.*—The recently introduced method of Sluder is designed to be done by one or two movements, somewhat after the manner of use of the old tonsillotome—a complete tonsillectomy, without any preliminary dissection. Sluder has lately revived the use of the tonsillotome as an instrument for tonsillectomy. The essential point of the operation is that with the aid of the Physick-Fahnestock-Mackenzie tonsillotome, with a dull blade, or some modification thereof, the tonsil is moved completely out of its normal bed in a forward and upward direction. Then with the aid of the eminence just above the mylohyoid line, produced by the last-formed molar tooth in its socket, the tonsil is pushed through the aperture of the guillotine. This bony projection he calls the alveolar eminence of the mandible. In children the tonsil is posterior to, and for the most part below the eminence, and appears further back and lower in maturity. The alveolar eminence is much more prominent in the young. Its position should be determined just before operating, by introducing the index-finger into the patient's mouth. To utilize the eminence in the removal of the tonsil, the tonsil must be moved forward and upward on to a motionless hard lump, as a solid, fixed and somewhat hemispherical convexity. The guillotine is placed over the tonsil at its base, the prominence of the eminence stuffs the tonsil through the aperture, the tip of the index-finger of the other hand being used to assist when necessary, as it sometimes is.

In operating, the tonsil is approached at an angle of approximately 45 degrees, which requires the shaft of the instrument to cross the mouth entirely from the opposite side. This necessitates the distal side of the shaft being applied to the tonsil. It is preferable to operate on the right tonsil with the right hand, and on the left tonsil with the left hand. For further details the operator is advised to consult Sluder's monograph.

This method, in the hands of its originators and those who have carefully studied it, seems to work out very well with a minimum of injury to the parts. I think in the hands of the beginner there is a tendency to do an incomplete operation, and I would not advise a physician who gets good results with his present technic to adopt it, although I recognize that in the hand of its originators it is a well-nigh perfect procedure. I have not yet felt called on to adopt it.

The removal of the tonsil by galvanocautery dissection is to be mentioned only to be condemned. While efficient, it is painful, and has no advantage over other methods.

HEMORRHAGE

For the prevention of hemorrhage, if it occurs, it is necessary to have ready some long hemostats, and to be prepared to tie a vessel if necessary. I do not believe, however, that it is necessary in every instance either to tie every bleeding point, or, as one of my friends invariably does, to suture the anterior and posterior pillars at the time of the operation. I have seen in the last year three cases of troublesome hemorrhage in adults, and none in children and young people among the cases on whom I and my colleague have operated. In two instances attachment of a hemostat a matter of two or three hours served to control the bleeding perfectly, and no other hemorrhage occurred. In the other the vessel was tied. As I said some years ago, in a paper which was commented on unfavorably by Cohen, I still find it very difficult to tie a bleeding point in the tonsillar fossa satisfactorily. One's instruments, fingers, mouth-gag, etc., seem to occupy too much room to do the work well, although I know it can be done, and have done it myself, but, as I said, with difficulty. I do not think we throat men become expert enough in tying vessels under difficulties. In several instances in which there was at the moment of operation a little profuse bleeding I have grasped the bleeding point with an artery forcep, given it two or three twists, and the bleeding has at once stopped. After the tonsil is removed with the capsule *in toto* the tendency is for the bleeding vessel to retract into the tissue and not to bleed. Except in adults who have had

1. The dissection is preferably begun at the lower half or two-thirds of the tonsil, as here the line of separation between tonsil and capsule is found without difficulty, and forms a good starting-point. The dissection is carried upward and then downward on the posterior marginal border.

repeated attacks of tonsillar inflammation and may, therefore, have fairly large vessels supplying the capsule of the tonsil, there is really not much danger to be apprehended from bleeding, but we must always be prepared to cope with it. The giving of chlorid or lactate of calcium for several days previous to the operation has seemed to me to be of some value, and I think that a small dose of morphin given hypodermically before the operation lessens the amount of mucous secretion and probably has some control over the question of bleeding. The multiplicity of instruments used for the tonsil operation proves that the question of technic is really simpler than it seems, since they can all be placed in three classes: first, the instrument to grasp the tonsil; second, the instrument to separate it from its attachments; and third, the instrument to complete the detachment from its external attachments.

The final test of any operation is complete enucleation with the least amount of time, trauma and risk. Let each individual operator endeavor to make himself perfect in the details of the method which appeals to him, and leave the other methods alone.

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ABSTRACT OF DISCUSSION

DR. H. W. LOEB, St. Louis: I so thoroughly agree with Dr. Richards that it is difficult for me to discuss the matter at all. The instrument he shows I would not use, but this is merely a matter of detail, which does not interfere with a full understanding of the principles of tonsillectomy. Not many years ago we decapitated the tonsils; lately we started to do tonsillectomies. Some valuable experience is gained after doing fifty or sixty more or less rudely performed tonsillectomies. Sluder deserves much credit for presenting an operation which can be done quickly. Dissection operations are done so much more rapidly than formerly that many are loath to give them up. The only objection to Sluder's operation is that it is so easy to learn; it is not easy to learn to do the first operation but after it has been done ten or twenty times it becomes an exceedingly easy matter. If Dr. Sheedy can operate on the tonsil in the way he describes and get such results, then the tonsils in New York differ from those in Missouri, and the work can be done more simply in New York than in Missouri.

DR. HOWARD F. PYFER, Norristown, Pa.: The results of tonsillectomy have not been entirely satisfactory. In Dr. Sheedy's series only 20 per cent. of the throats approach normal. His observations are confirmed by Stucky, Kafeman, Lewy, Shurly, Rabe, Stauffer, Getchell, Sawtell and Cocks. Failures in the operation are due, therefore, to using instruments such as guillotines, snares, scissors, angular knives and biting forceps mechanically instead of using our own technical, surgical skill. Instruments may do the work. Even Richards' finger dissection will sometimes succeed, but in small, flattened tonsils or in those bound down with cicatricial tissue following peritonsillar abscesses it is worse than an absolute failure, as it tears the superior constrictor and ribbons the pillars, producing dangerous hemorrhages and traumatism. We want a safe, sure, sane method.

What is good in Sheedy's operation? First, his method of anesthesia, in which he employs 1.5 per cent. bisulphate of quinin. The effect is instantaneous and without any constitutional disturbance or local irritation. Secondly, the use of a blunt-pointed but sharp tonsillar knife. (Fetterholf has shown the value of a superficial incision. Sheedy, however, distinctly states that his incision is intended only as a guide for the snare.) Thirdly, (if successful) the use of the tenaculum inserted as deeply as possible into the center of the gland, avoiding the capsule. Many tonsils, however, are not firm, the tenaculum tears out and then the eversion is a failure; this occurs even for Sheedy. The snare in Sheedy's cases works well, but, again, if there is any error of judgment or the wrong snare is used there is disaster. If one, from old love or traditional reverence must use the snare, Sheedy's method is far the safest and most efficacious. Sluder's guillotine, scissors, biting forceps or scalpel and snares all produce mutilation and are the source of hemorrhage.

Let me suggest a sane, safe, sure method, a mosaic of bright ideas from many sources. It can be used for any tonsil without careful selection, under local or general anesthesia, is successfully performed whether the tonsil is evertible or not, and whether loose, partially everted or bound down by adhesions or cicatricial tissue. No new instruments are necessary. First, use a cocain application followed by a quinin bisulphid injection, as recommended by Dr. Sheedy. Seize the tonsil with any favorite tenaculum or forceps and superficially incise the mucous membrane, thus outlining the tonsil. Dissect close to the capsule, finally arriving at the base; remove the small glands in addition to the tonsil just as tuberculous glands of the neck are followed. Catch any large bleeders with a hemostat, put in a stitch at the upper third of the pillars to increase the rapidity of healing; remove stitch in the morning.

Why prefer this method? Because it is always applicable, needs no perfected instruments, requires no exact technic or selection of certain classes of tonsils and, best of all, because there will be no mutilation or destruction of tissue, no loss of voice, no uncontrolled hemorrhages and, if each operation is conservatively, carefully and consistently performed, makes the surgeon more skilful. When the results to patient and surgeon are so invariably good, is not that a safe, sane, sure operation?

DR. WILLIAM L. BALLENGER, Chicago: The whole field of tonsillectomy has changed in the last fifteen years. I recommended the removal of the tonsil with the capsule intact and this recommendation has since been abused. When Dr. Sluder's operation came out, the other operation was abandoned. I am glad to know how to dissect the tonsil. I abandoned the operation I was doing because I had found a better and an easier way. I have used it for two or three years with the utmost satisfaction in from 70 to 72 per cent. of my cases. The dangers attending Dr. Sluder's operation I fail to meet in many cases. I avoid harming the anterior pillar. It is not so much the question of how you are going to remove the tonsil, but of whether or not you are going to remove it. The individual operator must be considered.

DR. OSCAR WILKINSON, Washington, D. C.: I prefer a triangular forceps. I catch the tonsil near its top, make sufficient traction on the tonsil to see its upper margin, but not sufficient to cause eversion, as that hides its superior outlines, make an incision through the mucous membrane only, and then use a dull dissector, merely scraping the tissue away from the capsule and never making a cut except in the primary incision. I simply peel out the tonsil and capsule. The edge of the knife is never out of view. When I have finished, there is only the smooth tonsillar fossae. The use of the dull dissector is very important and is of great aid. As a rule, I snare the lower part of the tonsil.

DR. JOHN O. MCREYNOLDS, Dallas, Tex.: During the past twenty-one years I have used practically every procedure recommended for the removal of the tonsils. We must all agree with Dr. Richards in his statement that any operation that gives perfect results and that is not objectionable in execution should be continued. At the same time, I feel that we have finally, and only within the last two years, arrived at a practical and perfect method for removing the tonsils; that is, the Sluder method. There are some minor differences in the method of operating. I do not pay attention to the alveolar eminence of the mandible. If I operate on the right side, I use the right hand. I pass the instrument across the oropharynx at an angle of 45 degrees, insinuating its distal end between the posterior pillar and the tonsil, lifting the tonsil upward and forward. The left thumb is passed into the mouth pressing against the anterior pillar, until I feel that there is nothing between the instrument and the thumb except the anterior pillar. With the right hand controlling the instrument, I crowd the blade forward, after getting a sure hold on the tonsil. With the other tonsil I use the left

hand, lifting the tonsil from its bed with the instrument, pushing with the thumb of the right hand against the anterior pillar, until the tonsil is pressed through the opening in the instrument, and then complete the section. In this way the tonsil with its capsule is removed absolutely. The operation can be done under local or general anesthesia. Any kind of general anesthesia will give equally good results, but hemorrhage cannot by any means be avoided. If hemorrhage does occur it must be arrested as it would be during any other surgical procedure. This method is applicable in nearly every case of tonsils in children, and in a very large percentage of those in adults.

DR. JOSEPH G. PARSONS, Sioux Falls, S. D.: Great credit should be given Dr. Sheedy for using quinin anesthesia. I have used it exclusively for local anesthesia in tonsil work for several years. The credit for the discovery of the use of quinin in tonsil work belongs to Dr. Brown of Minneapolis. Quinin is certainly a satisfactory local anesthetic. It has a distinct advantage in that the anesthesia is prolonged and much of the soreness which usually follows these operations is obviated. Again, it is a sort of insurance policy against hemorrhage, as good a one as you can ask for. It is inadvisable to use quinin stronger than in a 2 per cent. solution. This causes an outpouring of fibrin which is a guarantee against hemorrhage. I am glad to hear of its applicability to Dr. Sluder's method. General anesthesia is preferable for the successful application of his method, but it is not justifiable to use it in adults when the operation can be done successfully under local anesthesia.

DR. CHARLES FRANKLIN ADAMS, Trenton, N. J.: It is interesting to note what has been done in the past in the way of controlling hemorrhage. Until the sixteenth century the greatest loss on the field of battle was due to the deaths from hemorrhage. During that century Ambrose Paré instituted the use of the ligature; prior to his time surgeons were in the habit of staunching hemorrhage with the actual cautery, hot pitch and all kinds of styptics of the most cruel and barbarous nature being used. We seldom have deaths from infection, but we still have many deaths from hemorrhage following the removal of tonsils. The only sure way to prevent this is to tie the bleeding vessel, and there is no reason why we should not tie the vein as well as the artery. Tie every bleeding vessel in the throat. Much can be done by compression and torsion in cases of hemorrhage, but this is only temporary.

DR. C. ARMIN GUNDELACH, St. Louis: The statement of Dr. McReynolds that the alveolar eminence of the mandible is unimportant and may be ignored in the fulfilment of the Sluder operation prompts me to report my observations, which are quite in contrast to his. I feel that it is very important to take cognizance not only of the contour but also of the position, size, and shape of the alveolar eminence and of the slanting surface just posterior to the eminence. It has been my privilege and pleasure to see many operators when the tonsillectomies were performed with the Sluder method for the first time, and I have constantly found that failure to utilize the alveolar eminence invariably left a part of the tonsil behind. These facts were again demonstrated to me only this morning when I operated at the Eliot Memorial Hospital and then watched an operator try the method for the first time. In failing to utilize the alveolar eminence the instrument is usually kept too far back in the throat and the finger cannot be passed over the entire aperture of the guillotine, so that not only does the alveolar eminence fail to force the tonsil through the opening, but the finger is also prevented from completing the invagination of the tonsil. Furthermore, it is important to take cognizance of the eminence, for the instrument should not only be brought up to it but, once having come in contact with it, should also be held firmly in that position till the blade has descended and engaged the tonsil perfectly. The original instrument has been modified by Ballenger, Beck and others. I would suggest to the surgeon who is performing the Sluder operation for the first time that he use the original guillotine because its range of application is much wider and more simple than the variously modified forms; especially is this true, I think, of Dr. Ballenger's modification in the use of which the hand must be flexed to the utmost on the forearm, so losing the strength and security of a stiff wrist-joint in its normal position.

DR. C. F. WELTY, San Francisco: Applying a ligature to a bleeding vessel after tonsillectomy is very unsatisfactory, because of the danger of its slipping. I prefer to use a small aneurysm needle similar to the one Yankauer brought out for carrying my ligature. It is a means by which we can tie tightly and securely. After tonsillectomies it is also satisfactory to paint the cavity with tincture of iodin.

DR. G. SLUDER, St. Louis: I appreciate the honor you have done me and I am glad that my technic has been in the hands of friends. I believe, however, that it is well to adhere to whatever technic is employed, if you get good results. No one technic is satisfactory to all cases or to all men. It seems to me that my operation has the advantage of lightning-like execution. When occasion arises, I can remove tonsils, both faucial and throat, in twenty seconds under gas anesthesia, although I do not think it is always advisable to operate with such speed. The question of instruments I have omitted.

There is one constant factor with which we have to deal. When one man states that he cures over 50 per cent. of his cases, and another says that he cures over 70 per cent., and if you appeal to him and say that you can cure from 80 to 90 per cent. (and in my hands 99.6 per cent.) of all the tonsils, what will he say? In St. Louis one good man had twenty-four peritonsillar infiltrations in succession, and yet he placed his results at 99.8 per cent., which shows perfect execution.

One thing that always makes trouble is the establishment of an infiltrate about the tonsil. With some the sense of touch is so exact that a tonsil but one-quarter of an inch thick can be detected and removed and the cavities rarely exceed one eighth or usually one sixteenth of an inch. They are always troublesome and button-holing often results. There is one particular point that makes trouble, a small lobe high up at the junction. In attempting its removal vomiting or gagging efforts may result. Do not attack the alveolar eminence of the mandibulum, but take the upper one.

DR. A. M. CORWIN, Chicago: In my paper on the Sluder method read yesterday instead of to-day, by courtesy of the society, I took up the quasi objections to Dr. Sluder's operation and answered them. From the absence of all unfavorable criticism, I see that the method is evidently recognized by this body at its true value. I believe that it is here to stay. We perfect ourselves in one method and get satisfaction. A new principle or technic is suggested. We try it out and, if it is better, we adopt it. In this direction lies progress. A better method may yet be evolved, but ten years from now I believe that the Sluder principle will be most universally used.

Dr. Richards has practically covered the field of various methods in a broad way. But I think all methods can be classified more generically into four groups: (1) finger dissection—unsurgical and practically discarded; (2) cauterization of limited application; (3) pulling the tonsil from its bed with forceps, loosening by dull or sharp dissection and final severing with snare or cutting edge; (4) "Sluderization," which pushes the gland through a fenestra using the finger within the mouth and the apposed alveolar process.

In performing the Sluder operation we do not aim so much to get behind the tonsil with the fenestra as below and behind its lower pole. Pressure with the instrument upward, forward and outward keeps the gland in front of the fenestra, and counterpressure from the jaw and more especially from the operator's finger puts it through. This is the epoch making trick involved in the Sluder method.

DR. GEORGE L. RICHARDS, Fall River, Mass.: Throat specialists should know how to tie arteries better than they do; there are very few of us who have acquired the technic required for this procedure. I believe that the point in learning the technic of tonsillectomy is this: If the technic one is using is satisfactory, let him continue it, perfecting it in any details with which he is not satisfied. If he is not satisfied with present technic, let him pick out one that he wishes to learn, go to the man who has perfected that technic and learn from him how to do it.

V. 6 1913

THE RELATION OF OPHTHALMOLOGY TO DISEASES OF THE NOSE AND ITS ACCESSORY SINUSES

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The possibility of involvement of the orbit or its contents in diseases of the nose and accessory sinuses has been so well established from an anatomic point of view as to need no further proof. It is only necessary to study the investigations of Onodi, Birch-Hirschfeld and Loeb to be convinced of this fact. When considered from a clinical point of view, however, the frequency of the association is less constant than the anatomic investigations might lead one to suppose. While in many cases the causal connection is obvious, there are others in which a relation is difficult or indeed impossible to establish.

Diseases are excited in the eye through the nose by direct communication, in the orbit through the anatomic relations of the accessory sinuses, through nervous reflex action, and as a result of general toxemia. It is not always easy from subjective symptoms alone to determine whether the origin of the symptoms-complex is in the eye, the nose or the sinuses. The fact that an eye lesion coexists with a sinus infection is not in itself evidence enough to establish a causal relation. I have seen for instance, a case of tobacco-alcohol amblyopia, giving all the classical symptoms of retrobulbar neuritis, in a patient who had a chronic sinus involvement. Operation was advised, but postponed for various reasons. Alcohol and tobacco were discontinued and the patient made a good recovery. The operation on the sinus was not performed.

The enthusiasm manifested in the reports of cases following the publication of the work of Onodi lead, I believe, to conclusions that are not altogether warranted by the history of the cases reported.

A study of the reports of well-authenticated cases, however, leads to the conclusion that many of the diseases of the orbit or its contents can have their origin directly or indirectly in diseases of the nose, the accessory sinuses or the ethmoidal cells.

These diseases have been classified by McKay as follows:

1. Mucoceles of the sinuses.

2. Acute and chronic sinusitis with external signs of orbital cellulitis, orbital abscess, tumor growth, edema of the lid or dacryocystitis.

3. Sinusitis without external signs of orbital inflammation, but with ophthalmoscopic signs, as optic neuritis, neuroretinitis, retinal thrombosis or phlebitis; or without opthalmoscopic signs but with visual disturbance as scotoma, visual-field defects, ocular muscle paralysis or fifth nerve disturbance.

4. Cases in which the association of sinus disease has been asserted but questionable glaucoma, iritis, uveitis, keratitis, cataracts and vitreous opacities.

This classification should be modified possibly in some of its details, but the scheme in general is excellent. I shall not speak of the first two groups, but rather confine my remarks to the third and fourth.

The involvement of the optic nerve is said to be due to mechanical pressure by inflammatory exudation in the neighborhood of the sinuses, to distention of the sinus wall, to toxemia, or to occlusion of the central vessels, by embolism or thrombosis. The ophthalmoscopic examination may reveal hyperemia, a congestion with or without edema, a neuritis or a general atrophic process including the whole or part of the nerve. The patient may complain of dimness of vision in one or both eyes, vertigo, headache and other head symptoms. One or both eyes may be affected.

One of the most serious nerve affections depending on sinusitis is the retrobulbar neuritis, in which the papillomacular bundle becomes affected. The reasons often given for this early involvement (as shown by a central scotoma at first relative and later absolute) are pressure on the exposed portion of the particular bundle, pressure due to vascular changes in the central retinal artery, or toxemia. When one considers the position of the papillomacular bundle in the nerve and its relation to the central artery, it is difficult to see how pressure symptoms can play so important a part as is often assumed.

The macular bundle occupies the peripheral portion of the optic nerve for a distance of about 13 mm. from the globe. It then enters the nerve and soon becomes axial, where it remains throughout the remainder of its course.

The central artery enters the nerve at about the point at which the macular fibers begin to enter. At no time are the macular bundle and central artery in intimate relation.

Because of the proximity of the sinuses to the orbit, a general toxemia may be overlooked in our desire to establish a direct communication as the course of infection. The character of the infection or lack of resistance of the individual to the particular strain may be the cause of the optic nerve involvement, rather than anatomic proximity of the source and dominant symptoms of the infective process. This belief is substantiated by the statement of Onodi that there seems to be no relation between the sinus involved and the nerve affected. Is it not possible that chronicity rather than the location of the infection has most to do with the possibility of optic-nerve involvement, and that a toxemia rather than pressure is responsible for the eye symptoms? This might explain the oft-quoted saying: "The worst sinus cases seem to show the least eye involvement."

Whatever the source of the eye symptoms may be, some brilliant results have been obtained in retrobulbar neuritis through treatment of the sinuses, and no examination of these cases is complete which does not include that of the rhinologists.

The possibility of optic atrophy from posterior ethmoidal suppuration, or from pressure dependent on some sinus involvement, cannot be denied; but to establish a causal relation between the two is most difficult. I have never seen a case of optic atrophy, the origin of which I could prove was due to disease of the sinuses or ethmoidal cells.

That there should be wide variations in the experience of different observers is not to be wondered at when one considers the relation of the optic nerve as given by Onodi. He says:

> The optic nerve may come in relation with the sphenoidal sinus or ethmoidal cells, and the wall of the sphenoid may be very thick. The variation in thickness ranges between 1 and 12 mm. It is evident that even destruction of bone may occur in accessory sinuses and yet no visual disturbance result.

While there is no form of visual field contraction which can be considered pathognomonic, there is scarcely any variation in the field that may not be produced by

disease of the eye secondary to accessory sinus infection. The most common deviation from the normal occurs in cases of retrobulbar neuritis in irregular central scotomas with or without ophthalmoscopic findings. The attempt to establish a diagnosis of the sinus involved by the quadrant of the field affected has not been successfully established. That there is no uniformity of opinion in this whole subject is shown by the opposing statements of such men as Birch-Hirschfeld and Onodi. Birch-Hirschfeld asserts that central scotoma is an early and important symptom of tumor or suppuration in the posterior sinuses, due to damage to the papillomacular bundle, while Onodi has observed the absence of these phenomena in similar cases and also in one case of sarcoma filling up the nasal cavity. He also asserts that the visual fields are not narrowed in cases of ethmoidal cell suppuration. The more one studies clinical perimetry, the more one hesitates to base any diagnosis on the field findings alone.

The enlargement of the physiologic blind spot has received much attention recently as of diagnostic value in retrobulbar neuritis, but as yet our knowledge of the normal limits of the blind spot is too vague to warrant definite conclusions.

While paralysis of the extrinsic ocular muscles is of exceedingly rare occurrence in sinus involvement, paretic conditions may complicate mild cases of sinusitis, and become manifest as an insufficiency of one or more of the ocular muscles. The muscle itself or the nerve supplying it may become involved. The function of the muscle may be but slightly interfered with, and diplopia may not be present. The binocular vision may be reduced considerably, while the vision in either eye taken separately may be normal. The diminished binocular vision arises from the inability on the part of the patient to fuse the two images because of the muscle imbalance rather than from any deterioration in his visual acuity. As the impairment of muscular action becomes more marked, a diplopia will appear which, however, may have been present from the first in the extreme field of action of the muscle affected. Muscular insufficiencies are usually seen in cases showing acute inflammatory processes. Marked displacement of the globe may be found in cases in which the false position is assumed so gradually that the desire for monocular vision keeps the visual axes in their proper relation and the muscles adjust themselves to the changing position. Owing to their anatomic relationship the levator, the superior rectus, the superior oblique, the internal rectus, the inferior rectus and the inferior oblique are most exposed and therefore most often affected in sinusitis. The particular muscle involved may suggest the sinus affected, but as the lesion is often multiple too much reliance cannot be placed on this finding.

In certain cases the ocular symptoms may be the only demonstrable manifestation of the sinusitis, the results of nasal examination being negative as well as those obtained by transillumination and roentgenography. If all other etiologic possibilities can be reasonably eliminated, it is becoming common practice to operate on the sinuses in cases showing ocular symptoms which have been proved to exist, such as an infection of the orbit, or retrobulbar neuritis. On the other hand, in cases which show optic atrophy or true paralysis of the extrinsic muscles, confirmatory local symptoms should be present before surgery is resorted to.

I have had occasion to study only two cases of thrombosis of the central vein with reference to sinusitis, and in neither case was I able to establish a causal relation. The sinuses were not explored by operation, however, and as no satisfactory etiologic factor was determined in either case, the real source of trouble may have been overlooked.

In regard to the fourth group in our classification, cases in which the association of sinus disease has been asserted but is questionable, I should make some change. Included in this group are glaucoma, iritis, uveitis, keratitis, cataracts and vitreous opacities. I can see no reason why iritis and also uveitis should not result from an infection of the sinuses, just as they result from a similar infection in the alveolar process or the prostate.

The number of diseases remaining in the last group may be even less when their etiology is better known.

ABSTRACT OF DISCUSSION

DR. R. BISHOP CANFIELD, Ann Arbor, Mich.: I agree with Dr. Parker in many of the points he has made. As he said, it is important to understand that "the fact that an eye lesion coexists with the sinus infection is not in itself sufficient evidence for the establishment of a causal relationship." Enthusiastic believers in the intimate clinical relationship of the eye and accessory sinuses have found that many patients have been relieved from distressing headache and other symptoms of eye-strain after the refractive error has been corrected and the sinus operation performed. They have not always been able to tell to what procedure to attribute the result. I have often heard it stated that those who do not believe in the intimate clinical relationship between all the diseases of the eye and pathologic conditions in the accessory sinuses either were not able to make an exhaustive examination of the sinuses or did not care to do so. On the other hand, conservative men are now of the opinion that the most ardent supporters of the theory are being led away by their enthusiasm. The danger of operating on healthy accessory sinuses must not be overlooked. In my opinion, an operation on a healthy sinus is productive of more bad results than operation on the diseased sinus. I have under observation at present patients whose healthy sinuses have been operated on and whose condition is unsatisfactory; indeed, a great deal worse than before the sinuses were interfered with.

Indiscriminate exploration of the accessory sinuses without special indication and in the absence of corroborative Roentgen-ray and transillumination findings is to be deplored. Although there is no argument but that orbital suppuration and infection of the uveal tract are generally due to extension of disease from the accessory sinuses, trustworthy evidence to the effect that optic atrophy or glaucoma is dependent on sinus disease is, so far as I know, lacking. I do not believe that the sinus involvement can be determined by the character of the muscle insufficiency in view of the fact that some tremendous change in both sinuses and orbit does occur without the occurrence of any muscle anomaly. While I firmly believe in the occurrence of fundus lesions as the result of sinus disease, even as the result of so-called cryptic sinus disease, I am strongly of the opinion that operative exploration of the sinuses should be undertaken only after the most exhaustive and repeated clinical and laboratory examinations have been made. In this connection we must bear in mind that the orbit and its contents may become involved not only as the result of direct extension, but also as the result of toxemia or sepsis caused by the accessory sinus disease. The importance of this last group of cases must not be overlooked. In case exploration is decided on, it should be made in the most conservative manner possible until the extent of the sinus involvement has been learned, and it should be borne in mind always that operative interference with a healthy ethmoid, antrum, frontal or sphenoid may be followed by very unsatisfactory subjective results.

I have come to the conclusion that the average laryngologist knows less about the anatomy of the ethmoid than he does about the anatomy of any of the other accessory sinuses,

yet he attacks this region with impunity and ventures blindly into what is to him practically an unknown country, yet it is this region which is most intimately associated anatomically with the orbit and its contents.

Finally, while accessory sinus disease is certainly an important factor in the causation of fundus lesions, it is by no means so universal as one would be led to suppose by perusal of the literature.

DR. JOHN B. POTTS, Omaha, Neb.: I should like to report one case. Dr. Parker mentioned that an eye lesion was rare when there was sinus trouble. I have had one case that exemplifies that point nicely. A woman had a cold and a severe, persistent headache. It was thought that there was some involvement of the frontal sinus, the antrum or the ethmoid. Drainage seemed to be fairly good. The discharge was clear. Refraction seemed to cure the headaches except over one eye. The glasses were changed once, and atropin was used frequently, but with no effect on the persistent pain in the left eye. The middle turbinate of the left side seemed to be involved and enlarged. It seemed to me that the drainage from the ethmoid was good. I removed the turbinate and found a cellular condition. The pain in the left eye then ceased and has not returned since. What was the relation between the eye and the condition found?

DR. GEORGE E. SHAMBAUGH, Chicago: I should like to report in this connection three unusual cases of accessory sinus trouble that have come under my observation during the past year. The first was a case in which the diagnosis of a phlegmon of the orbit had been made, but in which an exploratory incision had failed to discover any pus. An examination of the nose showed a narrowing nasal passage on the affected side with a very slight alteration in the floor of the ethmoid. Tissue removed from this region demonstrated the presence of an epithelioma. The second case was that of a young man who had suffered for several years from severe headache and for a couple of years from severe hemorrhage from the nose. An examination of the eyes by Dr. Wilder disclosed nothing abnormal. The patient died from a severe hemorrhage from the nose. At necropsy a sarcoma was found involving the sphenoid sinus and the postethmoid cells. The third case was that of a woman in middle life who had suddenly lost the sight in one eye completely and in the other eye partially. Her trouble had lasted but a few weeks when I saw her. She could scarcely recognize fingers before the better-seeing eye. The eye symptoms had been preceded a couple of weeks by a severe cold in the head and headache. On examining the nose I could not make sure of any accessory sinus trouble. The history of the case led me to suspect an accessory sinus condition as the cause of the blindness. I opened the sphenoid sinuses and postethmoid cells. The operation had to be done under ether. I could not recognize any positive evidence of disease at the time of the operation. The patient recovered the sight completely in one eye and partially in the other.

DR. CORYDON G. DWIGHT, Madison, Wis.: The one point I wish to raise is the possibility of an association between sinus disease and brain-work. I have in mind a senior at the university who during his sophomore and junior years had trouble with his eyes and came to me for refraction. The glasses ordered gave him no relief whatever. I did not make a nasal examination then, but later I did and found a large middle turbinate and necrotic bone and involvement of the entire ethmoid. Many of these brain-users with accessory trouble complain more of their eyes.

At the same time I had a domestic, but one who did not do any brain-work. She had perisinusitis and absolutely no ocular symptoms.

In the clinical department at the University of Wisconsin Dr. Evans carried on a painstaking research and he reported that nearly 80 per cent. of the persons seeking treatment were either nasal or eye sufferers.

There is an acute ethmoidal trouble, which I believe was named by Dr. Sluder vacuum sinusitis, in which this condition exists. There is some hyperemia which produces symptoms and in many of these cases operation is not necessary. I believe that in 75 per cent. of the cases cited to-day the patient can be relieved by thorough washing out of the nose and keeping it clean. I do not think that patients must always be operated on. I believe in watching them, however, with great care. I have seen a great number of these cases and I am convinced that in many of them operation is not necessary. In some instances, however, it may be necessary to remove the turbinate. If there is a large middle turbinate, the region cannot be ventilated without operation.

DR. H. W. LOEB, St. Louis: A great deal of enthusiasm centers about the relation of the eye, ear and nose. My understanding of the subject was fairly well brought out in the anatomic specimens which were shown at the New York Academy of Medicine some years ago. In the great majority of cases the sphenoid is in relation with the optic nerve at its roof, and the opening of the sphenoid is in the middle third of the anterior wall, which allows a fair column of air between the level of whatever pus is present and the optic nerve. In five of the specimens presented, however, the optic nerve was either above or on the level of the orifice of the sphenoid. This naturally suggests the increased likelihood of infection in such cases, when pus is present.

With respect to the ethmoid, ordinarily the optic nerve passes close to the posterosuperior external angle of the last posterior ethmoid cell. This explains why, although suppurative inflammation of the ethmoid is common, it is seldom accompanied by disturbances of the optic nerve. In two instances, however, in which the ethmoid cells either displaced or surmounted the sphenoid the relation was different and the optic nerve ran along the external wall of the posterior ethmoid cell. Any disease of that particular cell would be far more likely to affect the optic nerve than the usual conditions.

The following case is of interest. A boy, aged 15, overgrown, hale and hearty, was taken with some affection of the eye. He noticed that his eyes lacrimated a great deal, that he had headaches and could not see well. After five weeks he was practically blind. The ophthalmologist referred him to a neurologist and the neurologist in turn referred him to me. There was swelling of the disk amounting to about 4 diopters on one side and 6 diopters on the other. There was a slight amount of vision on the left side. I found an acute ethmoiditis on both sides. The patient was sent to the hospital for operation. Forthwith the swelling decreased in one week, the vision increased, the headaches disappeared and the patient left the hospital entirely free from symptoms. He has been examined since and continues to have clear vision.

I think that in this type of case the posterior ethmoid cell extends posteriorly over the sphenoid to a greater extent. This patient had an acute ethmoiditis and the optic nerve was involved.

DR. F. P. EMERSON, Boston: I wish to relate two cases in which there were eye symptoms with apparently no evidences of any catarrhal process in the nasal mucosa, and yet that was the region in which the trouble was located. A woman, aged 38, had suffered from constant left supra-orbital headaches for twenty years and they occurred about twice a week. She was examined by the best men in the country. One treated her for faulty metabolism for one year. An eye man treated her eyes for a year. She was unable to find anyone who could alleviate her sufferings. Her headaches were very severe. She had clean nasal mucous membranes and no catarrh, but she did have nasal symptoms. Anything which produced nasal turgescence, such as sweeping, automobile riding or the menstrual period, would start this pain. These were distinct nasal symptoms. In looking into the nose you could see that she had what has been described so accurately by Dr. Sluder in writing of the conditions necessary to produce negative-pressure headaches, a septum deviated to the left side high up; the middle turbinate was close to the outer nasal wall and there was an enlargement or hyperplasia of the anterior end. Ewing's sign was present; that is, she was sensitive under the supra-orbital arch. I removed a portion of the turbinate and opened one or two

cells in the uncinate process to free the nasofrontal duct and she has had only two attacks of headache in two years.

The other case was that of a young woman who was supposed to have had a nervous breakdown and who had constantly recurring headaches, with pain through the right eyeball. She was constitutionally nervous, but it was thought that there was some other underlying cause for the headaches. The examination of the nose showed the same anatomic conditions presented in the other case. The middle turbinate was slightly enlarged and the region under the supra-orbital arch was very sensitive. The patient was cured by establishing drainage in the region of the nasofrontal duct.

I have seen several cases of the type Dr. Shambaugh describes in which there seemed to be a cellulitis, but in which nothing was found when an incision was made in the orbit. Here the trouble seemed to be in the posterior ethmoid region. In these cases, especially those described by Dr. Sluder, there are conditions which we overlook. If we have definite nasal symptoms and can find the anatomic conditions exactly as outlined, we shall be able to relieve these apparently intractable cases of headache or eye-pain.

DR. C. F. WELTY, San Francisco: I agree heartily with the criticism by Dr. Canfield. I have seen extensive disease of the sinus with few or no eye lesions. I wish to speak of the importance of the cerebellar pontine-angle tumors. This new work of Barronies makes possible an almost absolutely certain positive diagnosis. I have had four or five of these cases, a report of which will appear in the literature shortly.

I wish to speak also of an unusual condition, the association of cerebral tumors with mastoid conditions. I have had a case of this kind. It was one of acute mastoiditis with characteristic symptoms including facial paralysis and paralysis of the external rectus, which I did not recognize at the time. It was an ordinary case and operation was done. The paralysis was supposed to have been cured in a few days, but the patient did not recover. The man gave the history of having some growth in the mouth, which he had noticed three months previously and which was evidently postpharyngeal and hard. It filled the postnasal fossa. Four or five days after the mastoid operation the facial paralysis continued and the man complained of more headaches. I diagnosed brain abscess. The man would not submit to an operation and he died in a short time. Post mortem a sarcoma was found which extended from the postpharyngeal space of the pharynx, filling the entire middle fossa. I thought we were justified in assuming that this man had a brain abscess. I have never known nor read of such a tumor as that.

DR. GEORGE L. RICHARDS, Fall River, Mass.: We should be quite certain of the indications for operation before doing anything very extensive in the nose. There is such a thing as a proper conservatism. Not every patient is bettered by a nose operation, and we dwellers in the smaller cities where the end-results over a long period of time are known to us have to choose our operative cases with more care than the surgeon in the metropolitan center who never sees the patient after the operation and loses sight of him immediately after treatment is finished.

DR. J. C. BECK, Chicago: It is a wonder to me that no text-book speaks of any relationship between our specialty and neurology. I should like to report two cases. The first is a particularly interesting one. A young woman, a nurse, was referred to me by an ophthalmologist in Chicago. He said "I am sending to you a young woman who has a mental condition that is said to be catalepsy; please examine her and see what you find." I went over the case carefully. She had had a frontal sinus operation and a mastoid operation, and the hole in her nose was big enough to place a finger in, yet there were no evidences of suppuration, except that the parts were somewhat crusty. She appeared sallow, but not sickly. She was taken to the hospital and five different men made examinations of the blood, etc. I was asked to watch the patient. She would go to sleep at 5 a. m. and continue in a cataleptic state from which she could not be awakened; even sticking a pin in her would not awaken her. She had a slight twitching of the eyes. A diagnosis of hysteria was made. An external operation on the mastoid was done and she has since been absolutely normal, except for pain, which is now the only symptom of which she complains.

The other case is an important one from a neurologic point of view. This was a case of tumor in the region of the pontine cerebellar angle. It was a glioma which was observed by the best surgeons in New York, Philadelphia and Chicago. The patient was a man, 40 years old, who died from respiratory paralysis. A tumor was found post mortem at the base of the occipital lobe far inward. This patient had severe pain or headache which was referred to the front part of the head. One diagnosis made was syphilis of the brain; another doctor said it was possibly a tumor in the anterior portion of the brain. This shows how difficult it sometimes is to make a diagnosis. I believe that we should have a society in which neurologists, otologists and laryngologists can get together and discuss these matters so that we shall be able to understand these subjects better and to make better diagnoses.

DR. WILLIAM L. BALLENGER, Chicago: I have not heard it stated that we should verify our diagnoses by roentgenoscopy. In private work it is exceedingly rare to have a patient operated on without having from two to four pictures made to help verify the diagnosis and get an exact location of the sinus disease. By using the skiagram we are enabled to open the sinus at the exact point indicated in the plates.

DR. HOWARD F. PYFER, Norristown, Pa.: In small cities we are called on to do eye work as well as nose and throat work. My experience has been that the condition in the sinus which gives the patient trouble is due not to a large amount of pus present, but to a hyperplastic degeneration of the mucous membrane of the sinus. A radical operation is seldom necessary. Fracture of the middle turbinate toward the septum will often bring results. Many patients come to me for an ophthalmoscopic examination with the retinal veins congested and the disk hazy and injected. A rhinologic examination frequently shows that the diseased condition is in the nose.

Dr. Adams of Trenton states that many children complain of their eyes when the symptoms are produced by adenoids. Usually they have been sent by doctors, parents or teachers for glasses. A weak spherical or cylindrical glass gives partial relief, but Dr. Adams has shown that it is not the refraction that is at fault but the adenoids.

The State Hospital for the Insane is located at Norristown. An examination of the ears of the patients is frequently made and the resident physician sends immediately for the otologist if any ear complications are noticed. Despite these precautions and examinations, at necropsy a brain abscess otitic in origin is frequently discovered.

Dr. Dercum has said that a brain abscess can be present with no symptoms. Can he show us how a brain abscess may always be diagnosed?

DR. JOHN B. POTTS, Omaha, Neb.: I should like to second what Dr. Ballenger said regarding the use of the Roentgen ray as an aid in diagnosing these conditions. The condition of the eye is a troublesome one and closely related to the conditions found in the nose with a peculiar formation of corneal ulceration. Here we find intense pain and photophobia. These ulcerations will heal up in two or three days or a week, but they will recur. They are most likely to occur on the periphery of the cornea and not at the center. They recur a week or months apart and will recur in spite of all that may be done for them. An examination of the nose might disclose a deflection of the septum to one side and a pinching of the middle turbinate; an enlarged vomer, or ethmoid trouble, etc., may be found. In most of the cases there is not any distinct ethmoiditis. The correction of the deflected septum, however, will cause the ulcerations to clear up at once. This condition is usually associated with blepharitis. There is ciliary congestion. This is very painful to the patient; he cannot use his eyes at all and the condition will continue to recur unless this operative procedure is resorted to. I should like to cite a case referring to what Dr. Welty said about not seeing indications from the nose: A patient was

taken with acute ethmoiditis following a cold which he contracted in St. Louis. He came to Omaha. I found complete blindness in the right eye and that he could count the fingers with the left eye at not farther than 2 feet. The skiagram showed both sphenoids filled with pus. On opening both sides the condition at once cleared up.

DR. W. R. PARKER, Detroit: Dr. Shambaugh's cases illustrate well the point I wished to make. Two patients were without eye symptoms and one undoubtedly showed eye symptoms of sinus origin. I think these conditions are possible, but rare. I did not include in the paper inflammatory conditions of the conjunctiva or cellular tissue of the orbit, nor did I include reflex headaches, or cases of corneal ulcerations which depend on affections of the lacrimal apparatus. Cases of this kind are more common and well understood. My experience agrees with that of Dr. Dwight. In large student bodies we do meet cases of reflex headaches due to eye or nose affections. In these cases congestion of the mucous membrane of the nose, of the sinus and of the ethmoid cells may be found. A low-grade retinitis or a "fluffy chorioid" may also be found. You may refract and re-refract in these cases, but you will not cure them. If you relieve the nose and sinus condition, the patient will at once feel better. The trouble is not, it seems to me, due to the use of the brain, but rather to a relaxed condition of the mucous membrane incident to sedentary life. Get these persons out in the fresh air, with exercise and good food, and their symptoms will disappear.

THE MEASUREMENT OF AUDITORY ACUITY*

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The measurement of auditory acuteness is generally construed to be synonymous with the quantitative estimation of hearing and at first thought it would seem to be all that is implied by the term, and all that need be attempted in this method of examination. But a more comprehensive interpretation would compel us to include a qualitative as a really necessary part of a quantitative exploration of the auditory function, for in order that measurement of auditory acuity be complete, it should not be confined to the estimation of the power for the perception of one or even several tones, but the entire scale. According to the Helmholtz theory, each tone is separately perceived by its own particular cell and fibers which respond to this and no other. Therefore, the quantitative measurement in the usual restricted sense would test but a limited part of Corti's organ, leaving the rest unexplored.

Conversely, it is equally illogical to consider that the qualitative method can be completely divorced from the quantitative method of examination. Indeed, I maintain that actually it is impossible to do so, because we know, in both health and disease, the capacity to perceive tones of different pitch in different degrees of power, so that it can never be a question of perception only, but must also be a question of the amount of energy necessary to produce an excitation at the threshold of each and every tone.

At the two extremities of the scale points will be reached, of course, at which tone perception fails, no matter how powerful the stimulus, because the organ of Corti contains no elements which are capable of responding at all to sound-waves slower than twelve or sixteen or twenty, or more rapid than thirty or forty or fifty thousand vibrations per second, as the case may be, and, therefore, amplitude of the waves beyond such limits are of no concern.

Occasionally too, in the midst of the scale a hiatus will exist where perception for certain tones is completely lacking. But we believe that in reality the hiatuses are seldom absolute, but consist rather in a lessening of the auditory power for the tones in question, which might be still excited to auditory perception by sound-waves of adequate amplitude.

Let us consider specifically the most commonly used methods of examination of auditory capacity with a view to determining their value and their relative advantages and disadvantages.

SPEECH

The voice in speech is regarded by many authors as the ideal instrument for the measurement of hearing on the ground that we thereby test the auditory organ in what is really its chief function.

The singing voice of an average individual has a compass of about two octaves; the average speaking voice that of a few semi-tones. The normal audition, as we know, extends over a range of about eleven octaves. Speech, therefore, can test it in but a small proportion of its full extent. This being in the most used and perfected part of the cochlear organ, will therefore represent its most sensitive area.

If our purposes are merely practical, such consideration would recommend the speech test as a desirable method. But otologic science cannot be satisfied except by a more analytic method of mensuration. Moreover, the speech method of testing is of all the least accurate. As we have no means of graduating the intensity of the voice, we endeavor to start with a given vocal power and measure auditory acuity by varying the distance between patient and examiner.

But unfortunately such a method of testing audition is open to several sources of error. In the first place a uniform standard of intensity is not possible of realization. Voices differ not only in pitch, but also very materially in their clang-tint or quality, due to the presence of harmonics. Voices whose fundamental tones might be of the same intensity would, therefore, be heard differently according to their particular harmonic character.

It is not possible except in an approximate way to determine the dynamics of the voice. Our qualifying adjectives, "low," "soft," "loud," "ordinary conversational," as applied to speech, it must be admitted, constitute inexact expressions, which cannot serve as definite guides for intensity.

An ordinary conversational voice of one person may have a carrying power very different from the conversational voice of another, and even the same individual cannot be certain that it will be the same at different tests. It will not only fail, therefore, to serve as a standard comparable among different examiners, but even for the same examiner when testing the same case at different times.

In employing the voice as a means of testing hearing we may make use of phrases of sentences, of different words, of numbers, or of nonsense syllables.

The use of phrases or sentences is open to the obvious disadvantage that the individual examined will supplement his defect in hearing by his apperceptive faculties and guess what has been said by the context, aided by the emphasis and inflection of the speaker. Results, therefore, will depend greatly on the intelligence and education of the person being examined.

* Because of lack of space, this article is abbreviated in THE JOURNAL. The complete article appears in the Transactions of the Section and in the author's reprints.

The use of disparate words will lessen the errors arising in this way, but the apperceptive factor is still to be considered. Words will be grasped in accordance with the hearer's familiarity with them, and one's familiarity with different words will vary with age, training and education, profession and environment. Moreover, the auditory value of words differs considerably with the letters or elements of which they are made up. O. Wolfe, who made a detailed study of the elements of speech, came to some rather surprising conclusions with regard to the hearing distances for different letters. Vowels are heard much better than consonants, and, therefore, for a given word the vowel is the index of the range or hearing distance for that word. The extremes of distance are illustrated in the statement that a as in "father" is heard over 250 meters, while aspirate *h* is heard less than ten meters. Sibilant *s* could be heard 175 meters, while lingual *r* is heard only forty meters.

The use of numbers or nonsense syllables precludes some of the disadvantages of using words and sentences, but they still have the chief fault of all voice or speech tests, namely, the lack of a uniform and comparable standard of intensity.

Agreeing to an approximate standard of intensity as acceptable for our purpose, however, we still have to object to the method of measuring auditory acuity by varying the distance from the source of sound.

Andrews made experiments by which he obtained the percentage of accuracy of hearing of individuals placed at different ranges who had a series of specially selected lists of numbers spoken to them, and reached the conclusion that, while there is a general decrease in audibility as distance increases, yet the decrease is neither constant nor uniform.

These inequalities were attributed to the reflection or absorption of the sound-waves, which differed for different parts of the room. As it is impossible to avoid these acoustic phenomena in any ordinary testing-room, we must be prepared for such a source of error in all conclusions based on the correlation of auditory capacity with distance.

To mention finally another source of possible error in testing with a loud or even ordinary conversational voice, it must not be forgotten that, if one ear has still good hearing, it is impossible to eliminate it completely in the test. Though held tightly closed with the finger, or with a wad of cotton, it participates in the hearing.

There is the likelihood, therefore, of attributing to the ear tested a degree of audition which it does not really possess, but which has, in fact, been exercised by the other or normal ear.

WHISPER

The use of whisper for testing audition has advantages and partly avoids certain of the errors of conversational speech. It is more satisfactory for testing unilateral deafness as the unaffected ear is less likely to take part in the hearing. We can also come nearer with whispered speech to securing a uniform standard of intensity. This we do by following the instruction to utilize for our whisper test the residual air of the lungs after a forced expiration. It is likely too that the acoustic properties of the room do less to invalidate the estimation based on distance than in the case with loud speech. These are all advantages, relatively speaking, for the faults persist, though in less degree.

The absence of a uniform intensity is exemplified in the distance given as the normal hearing distance for whispered speech by different observers. They vary all the way from the estimate of Jasskau of ten or twelve meters for words and fourteen or fifteen meters for numbers, up to the estimate of thirty-five to forty meters given by Matte and Schultes.

We all realize how the result will be influenced by the acoustic properties of the room and its freedom or not from disturbing noises. In a room that looks on the street, the range of hearing will be materially reduced.

The quality and intensity of the constituent elements have been found to differ for whispered speech no less than for the loud voice. Quix, who has investigated this subject very thoroughly, has divided whispered speech into zones of pitch represented by the low, the high and the mixed zones. For the purpose of testing audition he finds it necessary to employ certain groups of words, which are what he calls isozonal and equiintensive, for he finds that one group differs considerably from another in the distance at which the words are heard by the same individual. So that it appears that while whispered speech has advantages over the ordinary conversational voice as a means of testing hearing, it still falls short of being an accurate and reliable method.

THE WATCH

With regard to the watch, the committee of the Bordeaux congress says:

> "Notwithstanding that it has not been possible to obtain from manufacturers a type giving about the same tone and having the same intensity, its employment is so convenient and the indications furnished relative to the auditory perception by the bony route so valuable that we believe it should be maintained as an acoumetric measure until we possess an instrument more exact.

Andrews says that one may hear the watch at a considerable range and yet be relatively deaf for speech and vice versa, for the ratio between the range of hearing for speech and that for a single tone of noise is not necessarily constant even in the same individual. This is an objection which can be equally urged against all tests which are confined to the estimation of hearing of a single tone or click or involve the perception of only a restricted portion of the basilar membrane.

In employing a watch as a test faulty technic may lead into erroneous results. A watch will give a sound of greater or less intensity depending on whether it is open or closed, nearly run down or newly wound up, whether it is held free or against the palm of the hand, and depending on its relation to the wall or other reflecting surfaces.

It has seemed to me that erroneous results attributable to the psychic element plays a greater rôle in the watch test than in any others. The watch click is a familiar one of such a rhythmic character that when it has just been heard, one can very easily imagine that he continues to hear it, thus deceiving both himself and the examiner. One will obtain results that differ greatly according to whether the watch is gradually removed from the ear until it attains the extreme range of hearing, or whether one follows the contrary course of beginning outside of the range and gradually approaching until the threshold of excitation is attained.

THE ACOUMETER

Many acoumeters have been designed, all aimed to present a method which might gain universal approval and adoption as a uniform method of auditory mensuration. The only one that has approached success in

V. 191

this ambition is that devised by Politzer. In this instrument one endeavors to assure a constant and uniform intensity by the regulated extent of the fall of the hammer on a metal cylinder. The Bordeaux committee on acoumetry recommends a little different method of using from that which is recommended by Politzer and which has been the method in general vogue. Instead of raising the striker at the end of the index finger to let it fall again, they advise letting it hang freely, causing the striker to take a swinging movement such as one uses in ringing a bell.

The objections to the acoumeter are very much the same as those to a watch, and one is liable to fall into many of the same pitfalls and errors in using it to measure audition.

Neither uniformity of intensity nor of pitch can be relied on in the different instruments which one finds in the shops, and besides many inaccuracies necessarily result, as I have pointed out in speaking of the speech tests, in which an attempt is made to correlate auditory power with the distance between the subject and the examiner. Furthermore, the click of the instrument is so loud that it is impossible to eliminate a normal and fairly well-hearing ear, and it is not applicable, therefore, in unilateral deafness.

TUNING-FORKS

The tuning-fork as an instrument of well-defined pitch and of relatively pure tone would seem to be the ideal one for testing the auditory functions. Its harmonics are few and far removed from the fundamental and generally die out quickly. They can be constructed to produce notes below the usual inferior limit and from there through the whole range upward to the highest perceptible tones. Nevertheless, by reason of the unlike intensity of forks of different pitch, we find that insurmountable difficulties arise in an attempt to apply them for either quantitatively or qualitatively testing audition.

In the deepest tones it is very difficult to get a satisfactory instrument. The vibration originating from within and without the prongs have opposite phases, which tend to produce interference. The forks, therefore, in their low pitches are so feeble that they must be held very near to the ear to be appreciated. As Politzer stated, the effect is to produce a sensation which may be mistaken for sound and give misleading results.

In employing tuning-forks to test the hearing, one attempts by various methods to get some basis for the estimation of the intensity. Three ways are possible: (1) to attempt to get a uniform initial intensity by using a uniform physical impact; (2) to calculate the intensity from the amplitude of the vibration; (3) to infer the intensity from the duration of the vibration.

One may either employ the maximal strike in setting a tuning-fork in vibration or secure a uniform intensity by a device such as that of Lucae. This consists in a hammer attached to the prong of the fork by means of a spring. It must be remembered in using tuning-forks that the more forcible the strike the greater the harmonics which tend to cover the fundamental tone. It is best that the fork be struck gently with a covered hammer and near the middle of the prong if one would eliminate as much as possible disturbing overtones. With regard to a uniform initial physical energy, it must be understood that even though this could be satisfactorily secured not much is gained, because the effect is different with each fork and the intensity must be estimated by varying the distance on counting the time of vibration, methods both of which are open to serious objection on their own account.

With regard to the estimation of sound intensity by means of amplitude, there are different opinions, and doubt is expressed of the validity of the claim of a simple relation existing between the two. It is generally asserted that sound energy is in direct proportion to the square of the amplitude, but this is denied by some authors. At any rate it is possible to estimate intensity from amplitude only in forks of low pitch; above the second octaves amplitudes are practically indeterminable. Ingenious optic methods of determining amplitudes have been devised by Gradenigo, Bonnier and Ostman, but they are similarly applicable only to forks of low pitch.

The third method of using tuning-forks, by duration of perceptible vibration, is the one most commonly employed. A serious arithmetical mistake is made, however, in stating auditory power in terms of duration, because the loss of intensity is not directly proportionate with time and is different for forks of different pitch.

Deep tuning-forks vibrate for a long time with a feeble intensity which diminishes slowly, while forks of high pitch vibrate a short time with great intensity at the beginning which rapidly diminishes. Bezold, Edelman, Gradenigo, Quix, Struycken, Dundas-Grant, Schmieglow and others have been at great pains to establish for the different forks their curves of decrement, but the calculations on which they are based are complex, and there is no agreement as to results.

After all, according to Quix, duration curves are determinable only for tuning-forks which test the sensitive zones of audition; "beyond these," he says, "one falls into the domain of hypothesis."

AUDIOMETERS

We have discussed all the methods of measuring auditory acuity which are in common usage, but, in addition to these, other methods have been proposed and certain special instruments devised for carrying them out. But none have so far been sufficiently free from all objection to gain general recognition or adoption.

The term "audiometer" has been arbitrarily applied to that type of apparatus which is placed at a fixed distance from the subject, and in which, instead of varying distances, certain special arrangements are provided for varying the intensity of the sound by increasing or lessening the physical energy which produces it. It is indispensable, of course, to be able to start with an intensity below the threshold of normal hearing and to raise it to reach the threshold of the very deaf, and it is essential that the energy be of a kind that can be readily measured in all its gradations.

Some of the earliest instruments designed to measure audition were of this type. Wolke, in 1802, employed a metal hammer falling from different heights on a sonorous metal plate, and the same principle is utilized in one of the latest audiometers, that of Toulouse and Vaschide, who substitute for the hammer drops of water, which are let fall from different heights.

Itard, in 1842, measured hearing by the strike of a small spherical mass, suspended like a pendulum, which could be made to strike a circle of copper from different points of removal. The distance of the swing was indicated on a dial which was therefore considered a measure of auditory power.

Again employing this principle an instrument has been very recently proposed by two Italians distinguished in this branch of otology—Stefanini and Gradenigo. The use of electricity to vary the physical energy

of the sound has been attempted by several authors, the most notable being the audiometer of Hughes and that of Seashore.

The chief difficulty with instruments of this type, if they are intended to secure universal adoption, is that it is not possible to realize uniformity in the different examples; their chief defect consists in the fact that they test but a restricted area of the cochlear field.

I have been experimenting recently at the government Bureau of Standards with a special machine which enables us to test the auditory function over a fairly wide area, namely, from 60 vibrations per second up to 3,000 per second, conducted to the ear by means of a sensitive telephone. An alternating current is passed through the primary of a variable mutual inductance whereby it can be readily varied in intensity. The different gradations of intensity can be accurately measured. I am thus able to produce sounds of varying intensity as well as of varying pitch fairly free from harmonics and thus combine, as we believe ought to be done, the qualitative and quantitative methods.

I have not progressed far enough as yet to feel justified in establishing any definite proposition, but I feel that such a method of testing audition embodies the right principles and will, if it can be simplified, become eventually the method of auditory mensuration *par excellence*.

CONCLUSIONS

From this general review of the chief methods of measuring auditory acuity, it is very evident that, as at present employed, they fall very short of the perfection greatly to be desired. They are both defective and deficient; no one of them is equal to all the demands of a complete acoumetry, and in what they attempt they are erroneous and easily lead to faulty interpretations.

The difficulties with which we have to contend are of two kinds, those which arise from the nature of the function being explored, and others connected with the various tests themselves and the manner in which they are conducted.

Strictly speaking we cannot really measure auditory sensation at all, because it is not a quantity divisible into equal units. We arrive at estimation of the power circuitously by measuring the energy employed in producing a given sensation, on the ground that auditory power is in inverse proportion to the intensity of the stimulus. We take as the criterion or standard of measure the lowest energy which we find is needed to produce just an appreciable perception of sound. This we call the liminal value, or from the standpoint of the ear, the threshold of excitation.

As we are obliged to depend on the interpretation and response of the subject, results will vary with degrees of intelligence and especially of aptness and attention. In testing with speech, for example, in which case the apperceptive faculties play an important rôle, education, training and environment greatly influence the response. Expectation and suggestion are psychologic factors which may vitiate the validity of tests otherwise carefully carried out, and in children it is necessary to eliminate as far as possible fear, nervousness and anxiety.

In some recent experiments I have shown that the ear by being continuously exposed to sounds readily shows fatigue which, of course, markedly lowers the threshold of excitation. This was found to occur with greater readiness with sounds at the two extremes of the trial scale, but especially with those of the upper limit. It has been proved that general fatigue of the body will lessen the acuity of the senses—including that of audition.

With regard to the tests themselves, there are many and various sources of error. These should be well understood and carefully borne in mind, if we would avoid the fallacies of too implicit faith in their accuracy and reliability. Of all the sources of error the one which is most carefully to be guarded against is that of depending on distance as a reliable means of estimating sound intensity. In the first place, if there is a relation existing at all it is not a simple one, as might be inferred from the common practice of expressing auditory acuity by a fraction in which the normal hearing distance is taken as a denominator and the distance at which it is heard as the numerator.

The fact is the intensity of sound diminishes with distance geometrically and not arithmetically. Suppose, therefore, a loud watch which can be heard normally at 100 inches is heard at only forty, individual hearing power is not correctly expressed as 40 per cent., but as 16 per cent.; while if the patient hears at only thirty inches his hearing acuity, instead of being 30 per cent., is really reduced to 9 per cent. But, unfortunately, when distance is used as a means of arriving at the intensity of the stimulus, the law of geometric diminution is upset by other disturbing factors. The absorption and reflection of sound-waves, the influence of extraneous noises and the participation of the other ear in tests made with loud sounds, especially when the latter are of high pitch, may each, singly or jointly, upset all calculations. Errors flowing from such causes enter especially into the commonly employed tests made with the tuning-fork, the watch, the acoumeter and speech and, therefore, seriously affect the validity of the results which are obtained.

The errors arising from the lack of a uniform initial intensity are encountered in those tests in which this is depended on. It is one of the drawbacks in the use of the human voice, the intensity of which can be only roughly gauged, and differs even with the choice of vowels and their arrangement, and also in the use of the tuning-forks whose intensity must be estimated by calculations that are complex and faulty. The presence of harmonics is a bad fault in many instruments used in acoumetry. They tend to cover the fundamental tones and so lead to erroneous conclusions.

Nearly all the tests which are in general use are open to the objection that they apply to only limited portions of the whole auditory field and cannot therefore be regarded as giving a complete measure of auditory power. It is this that explains the observation which has been frequently made that a person may seem to have a good hearing for the watch and at the same time have great difficulty in hearing speech.

Tests used with the idea of covering a great part of the auditory field, including the upper and lower limits —the so-called qualitative tests—are often applied without regard to the function of intensity. As I have pointed out, the limits of sound perception might be extended and apparent hiatus disappear by merely employing greater intensity. It might be useful to know the amount of intensity necessary to get the threshold of excitation for different notes of the scale.

A general survey of the subject of auditory mensuration leads to the inevitable conclusion that there is great need of a system that will meet the exacting requirements of otologic purposes.

The ideal instrument for testing audition, from an otologist's standpoint, would be one capable of produc-

ing all the notes of the scale, from the highest to the lowest, in pure tones free from harmonics and capable of giving these notes in intensities readily measurable and varying from a point below the threshold of excitation of the most sensitive hearing up to a degree necessary to awake sensation in persons suffering from the most profound deafness.

Moreover, in order that such an apparatus may deserve universal acceptation, it must be possible to construct as many others as are wanted of the same type, each constant for itself and precisely similar to the others. It is only then that we can hope to make it available for a uniform notation comparable among different observers.

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TONSILLECTOMY BY THE SLUDER METHOD

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It is a matter of pride to us that, though total removal of tonsils has been advocated and occasionally practiced for decades, laryngologists within the last dozen years have established enucleation in capsule as the operation of choice and clearly defined the indications for it. As a result of their effort, with its leadership in America, general practitioners and laymen alike are fast coming to demand this procedure. Quick to appreciate its value, they are equally alert to detect faulty work and ready to condemn its author.

We will all admit that enucleation is being well done by sharp or blunt dissection, by the aid of scissors or knives and forceps of various designs, in most of which combinations the snare figures prominently, and that the performance of tonsillectomy will by any method demand much study and practice. On the other hand, we must concede that there is much bungling work being done to discredit legitimate tonsil surgery and that the question of how tonsils can best be removed is still an open one.

It is opportune, therefore, to emphasize the value of an operation with which you are all more or less familiar, but which, in spite of its merits, has not yet received the recognition that is bound, I believe, to place it foremost of all methods for tonsil removal.

The large percentage of unsatisfactory results following the operation on the tonsils by use of the guillotine has done much to bring discredit on this operation. When, therefore, Dr. Sluder advocated the use of this instrument, modified in certain particulars, for the operation of tonsillectomy, most of the men doing this work were not prepared to accept offhand his statement regarding the effectiveness of his operation.

The clear descriptions of the Sluder technic make it unnecessary to dwell on it here. The essence of this new idea is that the tonsil, when scooped up by the ringed margin of a fenestra in one end of a stout shaft, introduced below and behind the gland, pressing it upward and forward and outward toward the rigid alveolar eminence of the jaw, can be put entirely through this opening, especially if counter-pressure is made on the tonsil with the finger inserted in the mouth and applied to the outside of the palatoglossus. When thus put through the fenestra the gland can be quickly separated from its attachments outside the capsule by guillotine, pushed as with Dr. Sluder's instrument and its modifications, or pulled as with the ring guillotine suggested recently by Dr. Jennings of St. Louis, or with Dr. Beck's snare or those of others, or severed by scissors or knife. One may do this operation by utilizing the ring in the handle of a strong pair of forceps and a snare.

In view of the simplicity of this method it seems strange that the guillotine was not used for tonsillectomy long ago.

I would emphasize several points of technic. First, it is essential to keep the shaft of the guillotine oblique across the buccopharyngeal cavity, with the ringed end pressed with sufficient force well below the lower extremity of the tonsil to insure the presence of the entire gland in front of the ring as it is brought upward and forward. Secondly, the importance of pushing the last vestige of the gland through the fenestra by judicious massage with the fingers is fundamental. To make sure of complete dissection — this is the key. Thirdly, the blade should not be too sharp, lest it unduly buttonhole the capsule and slice the tonsil itself. A sharp blade may be effectually blunted by taking off its edge with any metal object, care being taken that

A. M. Corwin's improved Sluder-Ballenger guillotine.

the knife margin accurately fits the ring against which it is to be thrust. Neither should the blade be too dull, since it requires undue leverage to force it home, and it does not dissect so well. The matter of hemorrhage, as between the use of sharp or blunt dissection, is, in our experience, negligible, bleeding in any event being controlled according to indications. Fourthly, after final pressure on the blade, the lingering attachments of mucous membrane and fibrous shreds should be severed with the finger so that the guillotine comes away easily without dragging on the tissues; and it always holds the removed tonsil in its grip, necessitating no use of forceps.

As an adjunct of this method, which has become an essential part of my technic for two years, I wish to commend the routine use of a pair of tonsil hemostats, of which there are several forms on the market. This one of my own design is simple, quickly adjustable and when in place is out of the way. The patient being asleep on his back, as soon as the first gland is removed, which takes but a few seconds, one of the hemostats is applied, immediately reducing bleeding to a minimum and allowing the removal of the opposite tonsil without

delay in a pharynx relatively bloodless. Twenty-five per cent. tincture of iron in glycerol (glycerin) has been used on the pad, and this alliance of stypsis and pressure has reduced the total loss of blood in my cases to a very small amount. In more than fifteen hundred tonsillectomies none have demanded attention from secondary hemorrhage, which is more than I can say for my previous work.

Incidentally, I find most satisfaction in anesthetizing all patients, young and old, with gas and oxygen in the hands of an expert, rather than with ether, chloroform and ethyl chlorid; in this regard I have been fortunate, and shall never again use any of the latter if the former can be had. This gives a greatly shortened period necessary for total manipulation, four minutes usually covering the whole procedure from the time the patient is wheeled into the operating-room until he is wheeled out on his way to bed. If needed, however, this anesthesia may be extended as long as desired.

The added security appeals to the laity as well as the surgeon. The value of the oxygen safety-valve is beyond question, and is a source of critical comfort to the operator in handling those occasional patients who do not take any anesthetic well. The relative absence of nausea and prostration after operation also commends this combination.

It is my mature judgment, based on large experience, that the Sluder method is applicable to all cases except those rare ones in which incomplete or mutilating measures or inflammatory processes have left the parts bound in a network of rigid, unyielding scars with little tonsil tissue present. Here judgment will advise the use of knife or cautery. Ordinary stumps and buried glands surrounded by adhesions, whether large or small, are easy victims to a skilful use of the method in question.

It has been asserted that no purely mechanical device can perform this dissection. This hypothetic objection is disproved by the experience of many who have done the Sluder operation extensively and are its ardent advocates.

A second objection, by Dr. Otto Freer, assumes that "to squash chronically septic tonsils through a powerful ring will necessarily force germs into the lymph-channels, especially if a concealed chronic peritonsillar abscess exists." He adds, however, that "considering the great amount of blunt tonsil enucleation being done, serious consequences are surprisingly rare, but are sure to be encountered in a large series." This is purely fanciful. Ample observation does not justify it. Such a dire effect has never occurred in my own extensive experience, nor has it been reported, so far as I know, by any of the dozens of expert operators who have used this measure. How unlikely such an occurrence is, even from a speculative point of view, follows from the fact that the capsule and inflammatory wall of a concealed chronic abscess are firm fibrous barriers, much more impenetrable than the tonsil itself, so that actually when the structures are pressed on from the outside, behind the anterior pillar, the adventitious contents of the organ tend to escape in the line of least resistance through the crypts into the faucial cavity. This is what takes place, the tonsil often being literally turned inside out.

Brains and experience are as much factors in the successful performance of the Sluder method as in any method. The size of the fenestra is important in this connection—the larger the tonsil, the more flabby or long in its vertical measurement, especially in adults, the more the advisability of employing the guillotine with the largest opening, which wholly eliminates even the imaginary danger of undue mechanical violence referred to.

Dr. Freer again has publicly called attention to the value of preserving the plica triangularis with which to line the muscular cavity from which the tonsil has been dissected. This structure, as convincingly pointed out by Dr. G. Fetterolf,[1] is a part of the tonsillar capsule. There seems no more reason for preserving it than for preserving the rest of the capsule, which to-day we aim to remove. Operative results of its removal sustain the wisdom of it. How many, in truth, operating by ordinary dissection, are preserving this plica and carefully stitching it to the surface of the pillars and superior constrictor in order to give this surface a covering of epithelium? Unless it is so held in place, how much good would it do to leave it?

Finally, much has been said about the cutting or tearing of the anterior pillar and dragging away of muscle fibers. I have here about a quart of miscellaneous tonsils for your inspection. If you find clinging to them an essential part of structures that should have been preserved, I should like to have this pointed out. I am only sorry that I have not the corresponding throats here from which they were taken in order to show the clean and perfect results that were attained clinically.

The Sluder guillotine has been dubbed by Dr. Freer a device of fear and incompetence to be used by those who cannot do scientific surgical dissection. Says the critic: "Those advocating such mechanisms always have in mind a typical tonsil for which the implement is constructed, and ignore the many changes produced in the tonsil and its surroundings by pathologic conditions, which make the operation of tonsillectomy one requiring great versatility." The attack, however, assumes that little judgment, skill or knowledge of anatomy is needed to apply the principles of the operation laid down by its discoverer. The facts are that many who are skilled in the employment of knife or scissors and snare have laid these by for this up-to-date guillotine as a better measure.

With scant courtesy toward its author, some have suggested that the instrument should be called the Mackenzie-Sluder, as if to hyphenate both names and honor, forgetting that no originality is claimed by Dr. Sluder for the guillotine idea, but rather for a totally new use of a very old device. By inference, no surgeon employing a scalpel should be entitled to fix his name unqualifiedly to any new operation, because, forsooth, the knife was first used by Sir Daggerous Flint, of the stone age.

It would not be surprising if the Sluder method were a failure in the hands of any one who relies for its performance on the cheap, grotesque imitations of Sluder's guillotine which are now on sale by certain instrument houses. There is no excuse for these misrepresentations in metal when the original was so carefully described by its author. There ought to be some method of rigid standardization among manufacturers of instruments marketed under definite titles.

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1. Fetterolf, G.: Anatomy and Relations of the Tonsil in the Hardened Body, Am. Jour. Med. Sc., 1912, cxliv, No. 1.

37 96

V. 191